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A Journal of Neurology

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~~PART I,~~ VOL. 41.

1918

THE INTERDEPENDENCE OF THE SYMPATHETIC AND CENTRAL NERVOUS SYSTEMS.[1]

BY DAVID ORR, M.D., AND LIEUTENANT-COLONEL ROWS, R.A.M.C.

EXPERIMENTS with bacterial poisons have demonstrated that the central nervous system can be infected by two paths, the lymph channels and the blood-stream [6]. The lymphatic system of the brain and spinal cord can be infected secondarily to a toxic or microbic invasion of the ascending lymph paths of cranial or spinal nerves, and the phenomena which arise are typically those of a primary inflammation of the fixed tissues. The examination of material from clinical cases, in which toxic absorption from septic foci had occurred along the lymph spaces of peripheral nerves to the nerve centres, confirmed the above results.

When attempts are made to infect the central nervous system by means of an intoxication of the blood-stream a totally different picture is found. In one series of experiments we introduced a capsule containing a broth culture of the *Staphylococcus pyogenes aureus* into the abdominal cavity of rabbits [8], with the result that definite areas of the spinal cord showed degeneration of the medullated fibres, viz., round the cord margin and on either side of the median septum of the posterior columns. These degenerations were associated with œdema, dilatation of the vessels, and hyaline thrombosis. Inflammatory phenomena amongst the fixed tissues were strikingly little in evidence. In another series of experiments [7] the capsule was placed in contact with the common carotid, and the examination of the brain revealed two types of lesion: (1) Coagulation necrosis of the nerve cells in the cornu ammonis, in the cerebral cortex, and in the amygdaloid nucleus; (2) softening in the stratum moleculare of the cornu ammonis. Here again the inflammatory phenomena were reduced to a minimum, except around the softening in the stratum moleculare of the cornu ammonis: but in this situation the proliferation of the fixed tissues was obviously secondary and dependent upon the irritative effects of the degenerative products in the surrounding tissues, and upon the reaction incidental to repair. Dilatation of the vessels, œdema, and hyaline thrombosis were present, and the above lesions clearly resulted from the consequent disturbance of the cerebral circulation.

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[1] This work has been carried on under a grant from the Board of Control.

These two types of infection, lymphogenous and hæmatogenous, are thus easily distinguished from each other. The former always gives rise to an inflammatory lesion which in both the peripheral nerves and in the central nervous system spreads by direct continuity. But the hæmatogenous lesions are much more difficult to explain, as the consideration of their anatomical distribution in the spinal cord and brain indicates very clearly that in addition to toxicity of the blood-stream there is another factor in play which determines where the toxin shall exhibit its primary effect.

This point is well illustrated by the experiments referred to above. When the abdominal cavity is infected a non-systemic lesion is found in the white matter of the spinal cord. Two areas are involved, the periphery of the cord and the posterior columns. In the cervical and lumbar regions the entire periphery of the cord is affected. In the dorsal cord this marginal degeneration is limited to the anterior and lateral parts. The degeneration in the posterior columns shows variations at different cord levels. In the lumbar cord it forms a small triangle round the hinder end of the postero-median septum with its base towards the cord margin. In the dorsal cord it forms an inverted V whose apex reaches the middle of the septum while its legs diverge towards the cord margin. In the cervical cord this degenerated area is larger than in the lower regions and forms a narrow column situated in each tract of Goll near the median septum and united to the marginal degeneration. The myelin lesion is maximal in the cervical, and minimal in the lumbar region. It is sharply confined to the above areas and consists in a primary degeneration of the sheath. With this myelin degeneration there are associated œdema of the cord, proliferation of the perivascular neuroglia, and dilatation of the vessels with hyaline thrombosis. In the grey matter the only morbid change is a very slight degree of chromatolysis of the nerve cells.

When the capsule filled with bacteria is placed in contact with the carotid sheath we find, as already mentioned, coagulation necrosis of nerve cells in the cortex, of the lamina ganglionaris of the cornu ammonis, and in the amygdaloid nucleus. There is also an ischæmic softening in the stratum moleculare of the cornu ammonis. Vascular dilatation and hyaline thrombosis are again a prominent feature. When the distribution of the lesions in both series of experiments is examined it is clear that they lie in those parts of the brain and spinal cord which derive their blood supply from the vessels of the pia-arachnoid. The implication of the cornu ammonis and the amygdaloid nucleus does not, as might appear at first sight, contradict this statement; for the lamina involuta of the former is penetrated by folia of the pia-arachnoid, while the amygdaloid nucleus is continuous with the grey matter of the temporal lobe. The affected areas therefore possess this point in common that they depend upon the pial vascular system for their nutrition; and it is this special anatomical distribution, to the total exclusion of the central areas, which inclines one to the view that a general intoxication is by no means a satisfactory explanation of the genesis of the lesions. A considerable weight of evidence can be advanced in favour of the view that the sympathetic nervous mechanism may be an important factor in determining at which points a toxin circulating in the blood shall exercise its primary action upon the central nervous system. Our authority for making this statement is derived from the study of certain non-systemic lesions in the cord, and from the anatomy, physiology, and embryology of the so-called autonomic system. Non-systemic lesions occurring in the spinal cord are characterized by atrophy of the myelin sheath and sclerosis. They appear to be dependent on some toxic condition of the blood-stream, which attacks the myelin sheath in certain definite areas by some obscure mechanism. We have studied such lesions in cases of visceral cancer and in Addison's disease; and on account of certain features which they possess in common with the subacute combined sclerosis of the cord, associated frequently with severe anæmia, we propose to include this group in the description.

If the spinal cord is examined in cases of visceral cancer a non-systemic lesion will be found which varies in degree from case to case. As an illustration let us examine in some detail the cord from a case of carcinoma of the pyloric end of the stomach. This case showed a more extensive lesion than is usually found, but in its general features and localization it was typical. The cord lesions in visceral cancer affect the posterior and lateral columns; the fine fibres of the grey matter are rarely implicated and only in advanced cases; and the morbid

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lesion consists in a slow atrophy and absorption of the myelin sheath accompanied by proliferation of the neuroglia. In the posterior columns the atrophic process varies in intensity in the cervical, dorsal, and lumbo-sacral regions. It is most marked, at all levels, round the postero-median septum and its area of maximum intensity changes frequently. Tracing it from above downward we find that from C1 to C3 it occupies a small area on either side of the posterior third of the septum. From C4 to C8, where the lesion is more pronounced than at any other level, it passes forward to occupy a more anterior position, until in C8 and in the upper six dorsal segments the areas immediately adjacent to the anterior third of the septum are most affected. From the level of D6 the area of atrophy progressively diminishes in size from above downwards and is shaped like an inverted V whose apex is directed towards the commissure and whose legs gradually diverge, on either side of the postero-median septum, towards the cord margin. The lesion at this level is of more recent date than that at a higher level. In shape it resembles closely the acute lesion found in the rabbit's cord after infection of the abdominal cavity. At the level of L1 there is some little degree of myelin atrophy confined to the fibres immediately surrounding the septal vessel and its branches, and from L3 downwards there is practically no change whatsoever.

The atrophic process accompanied by neuroglial proliferation begins round the postero-median septum, and progresses from within outwards. The lesion attains its maximum development between the fourth and eighth cervical segments, and in early cases we have observed that these and the highest dorsal segments are alone affected.

The lesion in the lateral columns is similar in nature to that of the sensory fibres but of less intensity. From C1 to C3 it is situated at the cord periphery and implicates both anterior and posterior cerebellar tracts. Below this level it occupies a more central position amongst the crossed pyramidal fibres and the lateral basis bundles. Similarly to the lesion in the posterior columns it diminishes in degree from above downwards and disappears almost entirely in the upper lumbar segments.

In Addison's disease the lesion in the spinal cord bears a remarkable resemblance to that in visceral cancer. It occupies the same position in the posterior columns. The morbid changes are more advanced in the cervical region and diminish from above downwards. They are of a more acute nature, however, and include more of the central portion of the posterior columns; while the histological picture presents certain differences indicative of a more profound morbid disturbance. Weigert's method demonstrates that the medullated sheaths in the affected areas are totally destroyed; and in sections stained by Marchi's osmic acid method one finds the adventitial lymph spaces of the vessels in the degenerate area filled with the products of altered myelin stained jet black. Evidently, in this case, absorption of the altered myelin has not kept pace with its destruction, and injury to the axis-cylinder has in all probability been a contributory factor to rapid disintegration. There is evidence in other areas of the cord of implication of the axis-cylinder. For example, there are scattered patches of sclerosis in the anterior and lateral regions of the dorsal cord, and one of these has interrupted many fibres of the right crossed pyramidal tract, causing a descending degeneration.

In the lateral portion of the cord there is some degeneration situated at the periphery and extending from the cervical to the lumbar region. This is different from what is found in the lateral area of the cord in cancer cases, but agrees with the results of those experiments in which the medullated fibres round the cord periphery showed primary degeneration. The fine fibres in the grey matter of the upper dorsal and the cervical regions are atrophied to some extent; in the lower dorsal and lumbar segments they are practically intact.

There is another condition in which the spinal cord shows a degeneration similar in character and distribution to the above, though of a more severe nature. Subacute combined sclerosis gives rise to a definite clinical picture, and is frequently associated with severe anæmia. The lesion is more intense than in cases of cancer and Addison's disease, and on that account tends to obscure the pathogenesis, but an analysis of the morbid features shows that apart from intensity the same morbid process is at work.

The portion of the cord most affected is the mid-dorsal, where there is marked destruction of the white fibres round the entire periphery, leaving the grey matter and the fibres in its immediate environment perfectly normal. The exogenous and endogenous fibres of the posterior columns are affected equally. Above the mid-dorsal region the lesion becomes more limited, that is to say, it is confined to the central portions of the posterior columns, to the two cerebellar tracts, and to the crossed pyramidal tracts. In the cervical region there are also scattered areas of degeneration, a favourite site for them being the direct pyramidal tract and the neighbouring bundles. The destructive lesion is found as high as the upper portion of the medulla; the upper limit of the pons, the internal capsule, and the cerebral cortex are perfectly normal. Below the mid-dorsal region the diffuse destructive lesion steadily diminishes in intensity, and in the lumbar region it is practically limited to the crossed pyramidal tract and to the posterior columns. Russell, Batten, and Collier [10] point out that we are obviously dealing here with a focal destructive lesion and system lesions dependent upon implication of ascending and descending tracts. The morbid process in its early stages is best seen at the margin of a degenerative patch. The myelin sheath first shows swelling, while the axis-cylinder remains normal. Later on, the sheath undergoes degeneration, and is absorbed, the axis-cylinder disappears, and sclerosis supervenes, attended by the ascending and descending degenerations mentioned above; but these are really secondary results. The grey matter, the spinal roots, and the posterior root ganglia are normal. The vessels in the affected area show thickening of their walls.

It is of importance to note that in most of these cases the degeneration of the posterior and lateral columns is most evident in the mid-thoracic region, and diminishes in degree and extent as the upper and lower limits of the cord are approached. The above-mentioned authors, in discussing the pathogenesis of these lesions, draw attention to their important observation that the distribution of the affection in the white matter is precisely that of the peripheral vessels, the pial supply, and point out how the areas supplied by the anterior median arteries—the anterior horns, adjacent white matter, and Clarke's column—escape.

We have described the above lesions as non-systemic, although it is obvious that in the posterior columns they bear a close resemblance to tabes dorsalis. There are cardinal differences, however, in the primary localization and further spread of the disease which separate it from a true tabes. The term pseudo-tabes might be employed on the clear understanding that the disease is not confined to the posterior columns, but is combined with sclerosis elsewhere.

If we review briefly the type of lesion in the three cases to which we have drawn attention, it is evident that we are dealing with three different degrees of one and the same pathological process. The situation of the degeneration is practically the same in all; especially in the posterior columns; and the late involvement of the fibres in the grey horns is a constant feature. In the cancer cases it is evident that the lesion is characterized by a gradual atrophy and absorption of the myelin sheath. So far as our experience goes, there is no evidence of axis-cylinder change, but the possibility of this occurring in other cases must be admitted. In the case of Addison's disease the pathogenetic agent is enabled to produce greater damage, for the lesion is not only more extensive, but we find the adventitial lymph sheaths in the posterior columns filled with fatty substance. In addition to this, isolated degenerative patches are found in which the axis-cylinders have suffered sufficient damage to cause a true Wallerian degeneration.

In the subacute combined sclerosis the morbid lesion is even more advanced. In the area of concentration, the mid-dorsal region, the axis-cylinders have been attacked, and the destruction has resulted in ascending and descending degenerations. Above and below the area the lesion is much less acute, and though its limits, above and below, are more extensive than in the cancer cases and in Addison's disease, it is similar in nature. The dissimilarity in extent might be reasonably explained by the fact that the morbid process has acted in a more acute fashion, and its effects have spread farther by diffusion of the noxious agent.

We have already mentioned that these non-systemic cord lesions appear to be caused by some toxic substance in the blood, but a general statement of this nature does not help us to explain the constant and peculiar situation of the lesions. They form a definite group—which, no doubt, includes more members than we have mentioned—whose characteristics make it reasonably certain that some special factor comes into operation in the pathogenesis. We assume that the myelin is attacked by a poison of some kind, and that this, if sufficiently strong, or in action for a sufficient period of time, can injure the axis-cylinder. We have no knowledge relating to the nature of the poison, but the lesions themselves raise an equally important question which is concerned with the mechanism by which these definite areas of the cord are invaded. There are insuperable difficulties in the way of our accepting a "general intoxication" theory without qualification. All the facts are against it. The distribution of the degeneration in the lateral and posterior columns, the localization of the degree of morbid change in the upper part of the cord, the gradual diminution of the atrophic change from above downwards until the lumbar level is reached, and the integrity of the grey matter so richly supplied by the anterior spinal arteries, all make the "general intoxication" theory unacceptable. It may be advanced that the affected areas are composed of fibres whose powers of resistance is less than that of others, but for this assumption we have no foundation in fact. Presumably the poison does not act unaided and indiscriminately, and there are reasons for surmising that the sympathetic nervous system is, in all likelihood, the intermediary in determining the localization of the lesions. We propose to bring forward evidence in support of this view, derived from recent work in connexion with the autonomic system, as many points in its anatomy, physiology, and embryology, along with some of its relationships to the endocrinic glands, seem to have a direct bearing on the argument before us. We have no accurate knowledge with regard to the part played by the sympathetic system in the causation of central lesions, but the probability of its action in this direction should not be denied for the present, in view of its intimate connexion with the spinal cord and vessels. Neuropathology in the past has provided many data of importance regarding nervous function, and the lesions under discussion, lying as they do, almost entirely within the realm of the thoracico-lumbar vasomotor system, suggest that the influence of this upon the central nervous system cannot be ignored. Our experiments affected special parts of the sympathetic system, and produced corresponding localized lesions.

The sympathetic nervous system, though intimately connected with the central nervous system, differs from it in many respects. It controls the visceral and involuntary functions of the body and has been termed by Langley the autonomic system. Its autonomy is only relative however; it has close anatomical and physiological relationships with the central nervous system.

Two ganglionated cords run along the ventral surface of the vertebræ from the atlas to the coccyx, segmentally interrupted by ganglia. Each is divided into cervical, thoracic, lumbar, and sacral portions. The cervical cord contains three ganglia, the thoracic twelve, while the lumbar and sacral portions have four or five each. The upper two or three ganglia of the thoracic cord are usually fused into one called the stellate ganglion.

These ganglia are termed vertebral or lateral: those situated more distally, for example in the cardiac, solar, and hypogastric plexuses, are called pre-vertebral or collateral, and small groups of cells lying more distally still in the course of the nerves before they reach their destination have received the name of terminal ganglia.

Each lateral ganglion is connected with the spinal cord by a grey ramus; only those corresponding to the spinal segments between D1 and L2 inclusive are connected to the cord by white rami also. These white rami, composed of medullated fibres, come from cells in the lateral horn of the cord (the intermedio-lateral tract) and conduct to the ganglionic cells of the lateral chain. The grey rami are not medullated, they are derived from the cells of the ganglionic chain which they connect with the peripheral organs.

Each white ramus sends off many collaterals and thus is connected with several lateral ganglia. The pre-vertebral ganglia are connected with the spinal cord by the splanchnic nerves, and are concerned entirely with internal visceral organs situated in the abdomen and pelvis.

The thoracico-lumbar connector nerves, that is the white rami communicantes, carry impulses to neurons in the vertebral ganglionic chain which supply (1) motor nerves for the muscles of blood-vessels over the whole body; (2) motor nerves for the musculature of all the sweat glands; (3) the whole pilo-motor system; (4) motor nerves for all unstriped muscle in connexion with structures derived from the segmental duct, that is, the ureter, uterus, and Fallopian tubes; (5) motor fibres to the sphincters of the intestine. The lateral, vertebral ganglia supply motor fibres to the blood-vessels of structures innervated by the segmental nerves, both spinal and cranial, including the central nervous system and the thoracic viscera. The collateral or pre-vertebral chain sends motor fibres to the blood-vessels of the abdominal and pelvic viscera.

There is an afferent system of sympathetic nerves which convey sensory impulses to the central nervous system. They are medullated and pass through the white rami communicantes, and have their trophic centre in the posterior root ganglia from which they run in the dorsal roots to the cord between the levels of D1 and L2.

The existence of vasomotor fibres in the brain has until comparatively recently been steadily denied. The action of adrenalin however has proved the presence of vaso-constrictors in the vessels of the brain, lungs, and heart, although it is admitted that their action is weak, and non-medullated sympathetic fibres have been observed to leave the main mass and proceed along the roots of the spinal cord which they reach by the posterior root especially. From these they pass into the cord membranes to supply the blood-vessels, but none enter the cord itself. Moreover, stimulation of the posterior roots has been observed to cause constriction of the blood-vessels of the dura mater.

The sympathetic ganglionic chain thus appears to be an extensive system of reflexes.[1] It is supplied with afferent and efferent fibres. From the level of D1 to L2 there is a connecting neuron within the central nervous system—the intermedio-lateral tract—which completes the arc between the afferent and efferent systems, and this fact, to which we shall return, seems to be of practical significance in connexion with the combined lesions in the cord already described. Another important point which must be kept in mind is that sympathetic action is dependent upon the supply of adrenalin. This point also shall receive further consideration.

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[1] We use the term "reflex" deliberately here.

It is not possible in view of recent additions to our knowledge of the sympathetic system to consider it as an independent entity. It is now known to be intimately connected both anatomically and physiologically with the ductless gland system. In fact, when we come to study the phylogenesis and ontogenesis of the vegetative system it is at once apparent that its relationship with the adrenal bodies is such that the development of each can hardly be considered separately. Phylogenetic and ontogenetic studies prove conclusively that the sympathetic nervous system, the adrenal bodies, and all chromaffin tissue must be considered as one in relation to the blood vascular system; and our reason for going into some detail in this direction is the importance of this relationship upon the cord lesions under discussion.

From animals low down in the scale to mammals, the close association of adrenal bodies with the sympathetic system has been recognized by many workers. In a primitive form of animal life, for example in the Petromyzon, there are two distinct series of bodies. One is represented by small lobulated structures in the wall of the posterior cardinal veins, of the renal arteries, and of the arteries dorsal to the kidneys. They project into the lumen of the vessels and consist of cylindrical or polyhedral cells containing granules which stain black with osmic acid. These are the cortical or inter-renal bodies. The other series—the chromaffin series—extends from the region of the second gill cleft to the tail, and each strip of tissue runs along the arteries and their branches. These structures bear the same relationship to the veins as the cortical bodies and correspond to the medulla of the suprarenals. These paired suprarenals lie in close contact with the ganglia of the sympathetic chain and contain large numbers of chromaffin cells. When we reach the Reptilia we find a closer relationship established between the cortical and medullary elements; and in birds it is still closer, the latter occupying the meshes of the former.

Mammals alone possess a true cortex and medulla, the latter completely surrounded as a rule. The cortex has the same structure as its homologues in the lower vertebrates, and consists of rounded groups or columns of cells containing fat-like granules which stain deeply with sudan III, scharlach red, and become blackened with osmic acid. The cells of the medulla have a great affinity for nuclear stains, and with chromium salts may assume any shade from a bright yellow to a dark brown. Cells exhibiting precisely this reaction have been found in the carotid body of mammals; and accessory chromaffin substance is found in any part of the body into which the sympathetic system extends, but especially in connexion with the abdominal sympathetic.

Now physiology teaches us that all chromaffin tissues yield adrenalin, or a similar chemical substance, and that the effects produced by this are almost all such as are produced by the sympathetic system. Comparative anatomy shows also that in addition to the medulla of the adrenal body there are numerous scattered bodies of the same nature in close relation to the sympathetic ganglia and nerves throughout the body. The adrenalin passes by the adrenal veins into the general circulation and assists in maintaining the tone of sympathetically innervated muscle and other tissue; and it is interesting to note in this connexion that the cells of the medulla are derived from the cells of the embryonic sympathetic system. Swale Vincent [11]—from whose comprehensive work we have borrowed extensively—affirms "that the cortex is derived from the mesoderm and the medulla from the same blastema as the sympathetic ganglia is now universally conceded." The intimate relationships between the vegetative nervous system and chromaffin tissue is therefore obvious.

If we look more closely into the development of the sympathetic system its intimacy with adrenal tissue from the earliest is clearly demonstrated.

There are two views regarding the genesis of the sympathetic nervous system, one that it is of mesodermal origin, the other that it is derived from the ectoderm. In a recent and important paper by Bruni [2] the whole question is revived and several facts of fundamental importance added. Observations in the higher vertebrates, and especially in birds, leave no doubt that sympathetic rudiments appear before there are any visible signs of rami communicantes connected with them. Bruni is of the opinion that the first limiting layer is derived from the mesoderm. Near the place where the segmental vessels leave the aorta and at the cranial extremity of the mesonephros small cellular mesodermal accumulations appear. In a short space of time these, at first arranged segmentally, are gathered into a continuous column and rapidly extend cranially and caudally, forming a dense protoplasmic trabecula. Such formations are particularly evident in the thoracic and abdominal regions. Two other facts deserve special notice. One is that in this mesodermal rudiment droplets of a substance develop possessing a strong affinity for hæmatoxylin. Secondly, simultaneously with the formation of these droplets, or shortly afterwards, neurocytes appear in the lateral branch of each spinal nerve—at least in the thoracic region—which accumulate at a point in the nerve least distant from the mesodermal rudiment and are projected towards it. Bruni is firmly of opinion that the mesodermal rudiment of the sympathetic is originally independent of the ectodermal portion, and adds that the former exerts an attraction on the ventral branch of the spinal nerve.

In a very short period of time the mesodermal and ectodermal rudiments become fused, and as development proceeds some of the trabeculæ (mesodermal) assume intimate relationships with the endothelium of capillaries lying laterally and ventrally to the aorta. The mesoderm then becomes rarefied, shows segmental swellings, and a few cells like posterior root ganglion cells. It is important to note at this stage that the mesodermal rudiment spreads laterally and in front of the aorta in the region where the cœliac and aortico-abdominal plexuses develop. A visceral ramus of each ventral spinal nerve now enters the mesodermal rudiment in the thoracic region: in the abdominal region part only enters, while the remainder expands in the region of the cœliac and abdominal plexuses to establish connexions with the mesodermal polynucleated protoplasmic trabeculæ.

From this stage onwards nerve fibres penetrate the trabecular mass freely, which thus becomes linked up by rami communicantes to the spinal nerves. With the advancement of development the polynucleated masses become more scanty, save in the region of the future cœliac and abdominal plexuses where they undergo a decided increase. These masses are permeated by nerve fasciculi, and are in intimate contact with the endothelium of the large capillaries. Still later in development the mesodermal rudiment, having attracted the fibres and cells of the spinal nerve destined to supply the true sympathetic elements, and having established connexions with the endothelium of the vessels, in part becomes transformed into a fibro-cellular cord, while a portion in the region of the cœliac and abdominal plexuses develops into the chromaffin formations which constitute the paraganglia and the medullary substance of the suprarenals.

There are some special points in connexion with the cervical sympathetic which deserve notice. Here the primary limiting layer gradually becomes less intimate with the spinal nerves and establishes connexions with the cranial nerves, especially the tenth, which in mammals contributes cellular elements early to the sympathetic cord. The superior and middle cervical ganglia are derived from a non-segmented cord, are not supplied with neurocytes by spinal nerves metamerically, but by elements which come up from the thoracic region and down from the ganglion nodosum of the vagus nerve. This is an important point, and Bruni mentions it in relation to the observations of His, Kuntz, and Abel, according to whom elements from the ninth and tenth cranial nerves supply supra-diaphragmatic plexuses of the sympathetic, especially the cardiac plexus and ganglia. Studies in comparative anatomy show that as we ascend the animal scale the components of the cervical ganglion tend to come progressively less from the spinal nerves and more from those of the sympathetic portion of the vagus.

An examination of the data obtained from a study of the sympathetic system provides us with certain facts which, if they do not afford a complete explanation of the lesions in the conditions mentioned above, at least are of sufficient importance to merit attention, and suggest a problem worthy of further investigation. There can be no doubt that the localization of these lesions in the central nervous system is not caused in a haphazard fashion in the course of a general intoxication. Such a definite localization as we have seen in the area of pial supply can be, in the cord at least, correlated with the spinal distribution of the thoracico-lumbar sympathetic system. This we have seen possesses central neurons (the intermedio-lateral tract) which send efferent fibres, preganglionic fibres, to the ganglionic chain, and receives afferent fibres, all, or nearly all, of which run into the posterior root ganglia—their trophic centre—and onwards into the cord by the posterior roots without entering into direct relationships with the sympathetic ganglia. The stimulus which these afferent fibres communicate must reach, therefore, the central nervous system before transmission to the periphery. We must conclude from this that in the sympathetic ganglionic chain there is no true reflex arc, or in other words that it cannot subserve reflex action without the intervention of the cerebro-spinal axis. But experiment has shown that in the sympathetic chain there are pseudo- or axonal-reflexes in which no afferent fibre is involved, and when a stimulus is applied to an axis-cylinder it can be transmitted by one of its collaterals to another cell or another ganglion as each preganglionic fibre sends collaterals to many ganglia.

Langley's work shows, however, that the sympathetic ganglia cannot maintain tone apart from the central nervous system, and the consensus of opinion seems to be that the ganglia merely act as reinforcing centres for stimuli from the central axis. But in the afferent and efferent systems of the spinal sympathetic centres and their connexions with the chain of ganglia we have all the elements of a true reflex arc, and the distribution of the spinal cord lesions is practically within the realm of this arc.

The involvement of the cervical segments does not contradict the argument, for, though the thoracico-lumbar mechanism lies within the limits of D1 and L2, it should be remembered that the lower half of the cervical cord is supplied by the ramus vertebralis composed of grey fibres derived from the stellate ganglion, which consists in a fusion of the first two or three thoracic ganglia. There are strong anatomical reasons, therefore, for the hypothesis that some disturbance of the sympathetic system may be held responsible for the localization of the atrophic and sclerotic changes, as they are only found in the vascular areas known to be under sympathetic control, i.e., those supplied by the pial vessels.

It is difficult to understand why the lower cervical and upper dorsal regions are most affected, and we have no explanation to offer. In all probability this problem, amongst others, involves an elucidation of the complex reflex paths in the sympathetic chain. Obviously these are not so direct as those in the peripheral spinal nerves, a point well exemplified by such common examples as the complex course of the dilator fibres of the pupil, the referred pain of visceral disease, and the unilateral malar flush so frequently seen in pneumonia. That the sympathetic system, however, comes into action in physiological and pathological conditions in the central nervous system seems to find additional support from the spinal cord lesion found in Addison's disease, whose syndrome is that of adrenalin inadequacy. Here we have muscular hypotonus and fall in blood-pressure generally, from which one can argue vaso-dilation in the pial supply of the cord.

We would suggest, therefore, that the lesions we have described are due to some interference with the thoracico-lumbar sympathetic reflex, caused possibly by pathological stimuli, or by the absence of substances which maintain its balance. This results in dilatation of the pial vessels whose permeability is thus increased. Hence the diffusion of noxious substances amongst the nerve fibres is facilitated, resulting in a progressive and ever-widening circle of atrophy and sclerosis.

If we are correct in the assumption that the organic cord lesions we have described are the result of some disturbance of the sympathetic system, then it would seem equally legitimate to suggest that certain so-called functional disturbances of the central nervous system may owe their origin, or if not that, their tendency to recurrence and permanency to the same mechanism; for, as we have seen, the sympathetic system is intimately linked up with the central nervous system, has no true autonomy, and there are anatomical paths which provide for a constant interaction between both.

Stimuli must be passing constantly from the sympathetic to the central nervous system, as there are special sense organs in the viscera and a definite sensory afferent path; and although these stimuli do not pass the threshold of consciousness normally, one cannot deny that they exercise physiological effects, knowing that under abnormal conditions they evoke pain. We must admit, therefore, that the stimuli from the sympathetic system exert an influence upon the brain under normal conditions, and play an important part in mental activity. In all probability stimuli coming from the vegetative organs to the brain, where they excite bodily sensations and instincts in consciousness, are of as great importance as those derived from the external world. And there is another point which must be taken into consideration. The relations which the sympathetic chain has with the vascular system and the ductless glands add very considerably to its functional importance in physiological and pathological conditions, so that in considering its influence on the central nervous system we have not only the effect produced by stimuli to take into account, but that through the vascular and endocrinic systems as well.

The precise relationship of the vascular system to the nerve-cells, and the mechanism by which the functional activity of these highly specialized elements is controlled, is still very obscure. Some modern workers are of opinion that the neuroglia plays some part in the process, and though the views advanced are not yet quite convincing, still with our knowledge so scanty as it is at present we must give them due consideration.

Achúcarro [1] considers that all the protoplasmatic neuroglia functionates as an interstitial gland which acts on the nerve elements and on the blood, contributing by means of special hormones to the endocrinic harmony of the organism. This view is based to a large extent on the presence of a foot-like process at the peripheral end of the neuroglia fibre which forms a connexion with the blood-vessels, and on certain embryological data.

The primitive neuroglia is represented by the ependyma, and, as development proceeds, the "neuroglial-ependymal" elements pass into the depth of the tissue. These embryonic neuroglial cells develop more rapidly in those ganglia which have acquired the greatest morphological and physiological importance. As the central nervous system thickens, and acquires a rich vascular network, the connexion between the neuroglia and blood-vessels becomes progressively more intimate, and Achúcarro advances this in support of his hypothesis that the neuroglia is a glandular vascular system. Cajal also is of opinion that there is a nutritive relationship between the neuroglia and the nerve-cell. When the nerve-cells are nourished from the ependymal space, they remain in that situation, and are unipolar; but when the vascular network is established, and the neuroglia becomes autonomous, then the neurons migrate, and become multipolar. It would appear from these authors that the neuroglia, under normal conditions, neutralizes noxious substances coming from the vessels, and destroys catabolic products from the nerve-cells, thus constituting a constant chemical rejuvenator, to a certain extent, in the substance of the nervous system. Achúcarro then pushes his theory still further, and applies it to the mechanism of the emotions. Granted, he says, that there is an indirect connexion between the nerve-cells and the vascular system, the neuroglia considered as an internal secretory gland could take part in the emotional mechanism. Recent researches, he continues, especially those of Cannon, have shown that in emotion not only is there a dynamic repercussion transmitted from the nerve elements, but there is also a true humoral repercussion expressed by an increase of glucosides and adrenalin in the blood, indicating hyperactivity of the adrenals. In this connexion Crile has drawn attention to an increased activity of the thyroid in emotion. Achúcarro thinks, therefore, that he is justified in advancing the theory that the protoplasmatic neuroglia, on account of its structure, relationships with the vascular system, and intimacy with the nerve-cells, can at certain times of nervous activity pour hormones into the blood which provoke other endocrinic manifestations with which emotionalism is associated.

Lugaro [3], criticizing Achúcarro's work, draws attention to its psycho-physiological nature in relation to the somatic humoral mechanism of the emotions. He points out, however, that emotion can be awakened purely dynamically through the nerve paths. Emotional phenomena take place with such rapidity that only nervous transmission from the brain to the periphery, and vice versa, could bring them into play. Were the emotional mechanism entirely dependent upon internal secretion, the emotion would occur slowly and last an excessive time. Lugaro notes that all the phenomena manifested during an emotion, or immediately afterwards, are not an integral part of its mechanism. It is conceivable that they could be for the most part purely collateral effects, devoid of any biological utility and perhaps, by their overflow, even damaging. He suggests the investigation of the relationships between certain conditions of disturbed hormonic equilibrium and the variations which they induce in the excitability of the nervous elements, especially those of the visceral system. Possibly for each type of disturbed equilibrium there may be an excessive facility or an abnormal inhibition of certain emotions. For example, there are the anxious apprehension in Graves's disease, and the phlegmatic character in hypothyroidism. An ideal affective equilibrium, ideal character, and a perfectly normal emotional state only exist with perfect hormonic equilibrium; and if lively emotions, especially when prolonged, disturb the hormonic balance they acquire greater importance inasmuch as an abnormal emotional state arises through disturbed humoral equilibrium. It is along such lines that Lugaro thinks we shall arrive at a knowledge of the physiology and pathology of the affections, and perhaps even find a rational somatic therapy of the dysthymiæ.

There is an interesting paper by Von Monakow [5] in which he enters a plea for the material basis of the sentiments and emotions. He is of opinion that the material basis of the sentiments ought to be regarded as chemical, not as anatomical or morphological. The chemistry of the blood and endocrinic glands comes into play as a varying and most important factor. The chemical composition of the blood is of cardinal importance for the sentiments; for sensation, motion, and orientation in time and space, it is—within physiological limits—of less importance. Even as low down in the animal scale as Vermes, there exists, in the cells of Leydig, an organ homologous with the endocrinic glands. In the human fœtus there are at the second month indications of the suprarenal capsule, which are intimately connected with the sympathetic ganglia; and the extreme sensitiveness of the sympathetic to pharmaco-dynamic action has been demonstrated by adrenalin, pituitrin, cocaine, nicotine, atropine, &c. Cannon and

Asher have shown that psychic excitement determines, through the sympathetic paths, a hypersecretion of adrenalin into the blood. The internal secretory glands, therefore, influence the nervous system in general, and the sympathetic in particular, and so provide a diffuse mechanism which regulates the vegetative or instinctive life of the organism. Thus the way is paved for elective chemical actions to create, on the exhibition of special stimuli, a favourable or inhibitory influence, and determine, amongst a conflict of stimuli, the appearance of a certain sentiment. Von Monakow then proceeds to discuss the epochal development of the sentiments in man and his conclusions are of the greatest interest. He says that, broadly speaking, the evolution of the sentiments cannot be placed in clear relationship with the phases of anatomical development. All we are justified in saying is that the primordial sentiments ought to have a more solid organic basis than those of later growth, and therefore under morbid conditions their resistance to the tendency to dissolution is greater. He supports the psycho-analytical school who ascribe great importance to the psychogenetic factor in the functional psychoses, neurasthenia, obsessions, and hysteria, but tempers their views by an unqualified admission of somatic factors. While he readily grants the frequent incidence of degeneration as a predisposing factor, great stress is laid upon the biochemical, organic, or somatic factor, that is to say, one absolutely apsychic. For example, moral trauma, severe psychic conflict, unsatisfied strong instinctive tendencies, disturb, through the sympathetic system, the endocrinic glands; and thus changes arise in the blood, which, combined with prolonged psychic stimuli, create nervous instability, resulting in neurasthenia, hysteria, phobias, and hypochondriasis.

"Evidently then," as Lugaro remarks in his criticism [4], "the nervous system and the endocrinic glands act as one under certain circumstances and constitute the basis of many changes in normal psychic life; both are naturally more operative in certain pathological states, which psychologists have tried to explain by fantastic hypotheses. Between moral trauma, which arises from a psychic cause and its psychopathic effects there are a series of phenomena which no doubt come under the term 'unconscious.' But the 'unconscious' is not necessarily that of the psycho-analyst, psychic unconsciousness; it is conceivably the result of chemical phenomena as unconscious and apsychic as any other."

The psycho-neuroses of the war provide an ample field for the application of all the above data, and demonstrate conclusively how disturbance of the sympathetic mechanism, of the endocrinic glands, and morbid emotional conditions, combine to bring about pathological psychic and nervous phenomena. Doubtless these three factors do not act separately. From their physiological intimacy we can hardly escape from the conclusion that all three act together.

Pathogenesis is invariably a difficult problem and especially so in the case of psychoneuroses. As Pighini [9] points out in his discussion of the clinical symptoms and pathogenesis of emotional psychoneuroses, we still know little of normal psychic processes, and of their connexion with other organic processes in the body. The physiology of pleasure, pain, and of the emotions generally shows us, however, that these psychic states are associated constantly with somatic repercussions which are maintained especially by the sympathetic nervous system; but much obscurity exists regarding the manner and path of conduction by which these come about. Pighini is an adherent of Bechterew's view, that in the cortex there are so many successive sensory-motor regions with psychic functions. Each of these regions consists in a receptive centre which is in intimate functional and anatomical connexion with a neighbouring efferent centre, thus forming an organic neuropsychic unity, e.g., optico-motor, acustico-motor, tactile-motor. Sympathetic centres are intimately connected with the above and take part in all psychic and psycho-sensorial processes, and thus it is easily conceivable how the sympathetic system becomes involved in disturbed neuropsychic activity by direct transmission from the original site of injury in the sensory motor cortex. For example, the hyperexcitability of the cortical visual or auditory zones, induced by shock, may be diffused to the limiting efferent regions and onwards to the inferior centres in the mesencephalon, medulla, and cord. Hence there arise dilatation of the pupils, nystagmus, cardio-vascular and vaso-motor phenomena, and other symptoms.

The more extensive the neuropsychic zone excited by emotional trauma is, the more accentuated and complex is the morbid repercussion in the sympathetic sphere. These are always more or less accentuated, and are an integral part of the syndrome of traumatic psychoneurosis. The purely psychic symptoms of stupor, acute confusion, hallucinatory delirium, excitement, and exaggerated emotional tone come on as a rule before the somatic symptoms due to involvement of the sympathetic and endocrinic systems; and Pighini is of opinion that this implication assumes, secondarily, the preponderating part in the morbid process and prevents recovery of the disturbed central function.

The somatic symptoms, therefore, are worthy of the most minute examination, seeing that they may prove to be the key to the correct interpretation of the morbid state.

In regard to the sympathetic system the most prevalent signs are those of hypofunction. It must be granted, however, that it is often a difficult matter to decide whether the disturbance is due to hypofunction of one system or hyperfunction of its antagonist. For example, in a case presenting dilatation of the pupil and tachycardia one has to determine whether these depend upon heightened tone of the sympathetic or diminished tone of the antagonist third or tenth nerves. Pharyngeal and laryngeal anæsthesia, paresis of the vocal cords, tachycardia, asthma, frequent vomiting, the absence of the oculo-cardiac reflex and its inversion, all point to vagus hypotonia; while bladder trouble points to involvement of the pelvic nerve.

There are other symptoms indicating involvement of that part of the sympathetic in close relationship with the endocrinic glands. These are dermographism, hyperidrosis, epigastric impulse from the aorta, exophthalmic goitre, and erythromelalgia. Tremor and clonism accompanied by tachycardia, marked asthma, slight hyperthermia, hyperidrosis, and psychic hyperirritability are common. Pighini attributes those symptoms to endocrinic disturbance, and is inclined to give preference to an altered functional state of the thyroid gland. He has found that the thyroid gland reacts readily to sympathetico-cortical trauma, and, having undergone change in function, in turn it induces morbid complications amongst other units of the complex endocrino-sympathetic system. This inevitably leads to a reduction of hormone influence and consequently autonomic hypotonia, whose symptoms amongst others are tremor, Addison's disease, Raynaud's disease, emotional hyperidrosis, diabetes mellitus, and angioneurotic eruptions. All these conditions have been observed to follow violent mental suffering.

There are certain neurological conditions incidental to war wounds, such as trophic disturbance in limbs, osseous and muscular atrophy, hypertrichosis, hyperidrosis, œdema, hypothermia, anæsthesia, paræsthesia, paresis, causalgic pain, and other symptoms which are not referable to lesions of the sensory or motor nerves, but rather to concomitant sympathetic disturbance. These symptoms according to Pighini are true traumatic tropho-neuroses of reflex origin. It would seem therefore that the function and conductility of sensory and motor nerves can be injured by vasomotor changes.

From all the above it is quite evident that there is sufficient justification for the suggestion that the sympathetic nervous system, though in physiological control of the involuntary functions, plays a much greater part in central nervous processes than has been previously admitted. However one may look at the problem, certain facts emerge which prove the close interdependence of the vegetative and central nervous systems.

The situation of the experimental lesions in the cord and brain noted above supply us with this fact, that these are the result of disordered nutrition and occur in the regions of the central nervous system whose vessels are under sympathetic control, that is, in the area of pial supply; and the remarkable distribution of the spinal non-systemic lesions in the three clinical entities considered affords additional evidence of the operation of the sympathetic factor. At least one must attach no small significance to this fact, that these lesions in the cord occur within, and attain their maximum intensity in the upper part of, the spinal area from which the white rami are projected to the sympathetic chain, and pass the bounds of this area to a slight extent only. But when we come to study the embryology of the sympathetic system and its intimacy with the endocrinic glands, and their joint physiological effects upon the central nervous system and *vice versa*, our prior arbitrary division of these into separate organs —in the physiological sense—becomes manifestly absurd; and the clinical evidence substantiates this view.

It is apparent therefore that in nervous diseases, and in those especially which concern the cerebrum, we must in future study not only the more prominent symptoms dependent upon disturbed function of the central organ, but also the collateral symptoms arising from disturbance of all the peripheral organs intimately connected with it. Anatomy, physiology, embryology, psychology and clinicial neurology indicate clearly that the problem cannot be solved by a narrower programme.

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CONDUCTION IN PERIPHERAL NERVE AND IN THE CENTRAL NERVOUS SYSTEM.

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INTRODUCTION.

WITHIN recent years the process of excitation and conduction in the peripheral nerve-fibre has been analysed with considerable accuracy and our conception of the nervous impulse has become far more definite. This analysis is inseparably connected with the name of Keith Lucas, and it is grievous to speculate how far he would have extended it if he had lived. The results so far attained may be considered from two distinct points of view. In the first place they represent the groundwork of an attempt to express a function of the living cell in terms of physics and chemistry. They are studies in cell physiology rather than in neurology, and though the particular function investigated happens to be that of nervous conduction, this function was chosen only because it happens to be very highly developed in certain specialized cells and therefore susceptible to much more accurate measurements than other functions such as those of assimilation or secretion. The investigation of the temperature coefficients of the different phenomena in nerve [5], [27], the characteristics of the electric current required to excite [29], the relation of the size of the impulse to the rate of conduction [6], [21], and similar problems are all directly concerned with this aim and their results form a body of evidence on which any theory of nervous activity must be judged. They have already led to theoretical results of some interest [32], but any advance on these lines must needs be a slow business, since it is limited by our very imperfect knowledge of colloidal phenomena and, although this need not deter us, it may turn out in the end that the vitalists are in the right when they assert the impossibility of describing vital processes in terms of the laws of mechanics.

But the analysis of conduction and excitation in the nerve-fibre may be considered from an entirely different point of view, that of the neurologist who is concerned not with the physics and chemistry of the nervous impulse but with the mechanism by which the impulse is made to serve a useful purpose in the body, the mechanism of the reflex arc and of the central nervous system in general. Unless we know the chief characteristics of the nervous impulse in simple conducting tissues, such as the peripheral nerve-fibre, it is waste of time to speculate on the nature of inhibition, summation, and all the other modifications of nervous activity which make up the work of the central nervous system. In this sense a knowledge of the behaviour of the impulse in a single nerve-fibre must form the basis of all neurology which is not concerned exclusively with the anatomical and functional arrangement of different tracts and cell stations in the cord and brain.

As will be seen, the analysis of peripheral conduction reveals several principles which are of direct importance to neurology. These principles have been outlined in the following article, which is little more than a summary of Keith Lucas's work on the nervous impulse [34]. It must be understood that the extension of our knowledge of peripheral conduction to cover the case of the central nervous system as well can only be justified as a forecast which may need a good deal of modification when the evidence is more complete, but it is at least a forecast which may suggest future lines of attack.

CHAPTER I.—THE IDENTITY OF THE IMPULSE IN THE NERVE-MUSCLE PREPARATION AND IN THE INTACT NERVOUS SYSTEM.

As most of the evidence has been derived from experiments on the classic sciatic-gastrocnemius preparation of the frog, it is as well at the outset to consider how far we are justified in assuming that the impulses set up in an isolated nerve by electric stimuli applied to the nerve trunk are identical with the impulses passing in or out of the central nervous system in the intact animal. The point is so important that it merits discussion in some detail.

Clearly, if what we call the nervous impulse in an isolated muscle-nerve preparation is an artificial effect quite unlike anything that happens in life its investigation will not help us much. As a matter of fact this extreme position is seldom advocated and there is abundant evidence that impulses identical with those studied in isolated nerve do form a large proportion of the impulses which occur under normal conditions in the body. The electrical stimulation of motor and sensory nerves in the living subject produces effects which differ in no way from those due to normal reflex activity; the rates of conduction are the same and the muscle gives the same oscillating electric response. Much of this evidence has been discussed at length by Piper [37], but the most conclusive proof comes from the recent experiments of Forbes and Gregg [18]. In these experiments it was possible to photograph the changes of potential in a motor nerve forming part of a reflex arc and called into activity by stimulating the appropriate sensory nerve. Taking into account the slight differences introduced by variations in the length of the conducting path, &c., it was found that the records of reflex activity showed electric responses which did not differ in any way from the electric responses obtained by direct stimulation of the isolated nerve. Thus we can scarcely doubt that the active contractions in a reflex arc are called up by nervous impulses of the same nature as those which form the subject matter of the classical muscle and nerve physiology. But it may reasonably be questioned whether there are not other forms of conduction in the intact nervous system quite unlike the form which can be studied in isolated nerve. It must be admitted at once that we know very little of the nature of conduction in non-medullated nerve and we must be content to regard the action of the autonomic system as outside the scope of the present inquiry. However, the real difficulty arises from the fact that we do not know whether the tonic postural contraction of voluntary muscle is controlled by the same nervous mechanism as that which regulates the active phasic contraction. The evidence on this subject is still very inconclusive. Undoubtedly there does exist in certain muscles, e.g., those operating the claw of the crayfish, a tonic mechanism which enables the muscle to remain shortened for long periods without appreciable loss of energy. This steady contraction appears to be entirely different from the tetanus produced by repeated stimulation of a nerve leading to the striated muscle of a vertebrate. Whether the same type of mechanism comes into play in such conditions as decerebrate rigidity, or indeed in all forms of postural contraction, is still an open question. Many attempts have been made to show that voluntary muscle contains two distinct contractile mechanisms for the twitch and for the tonic contraction supplied by two sets of nerves, medullated and non-medullated, or by two forms of nervous impulse. If this is so it is clear that the production (and inhibition) of tone must be due to a mechanism quite distinct from that studied in the muscle-nerve preparation. The theory owes most of its development to Bottazzi [15], who identifies the two contractile mechanisms with the sarcostyles and sarcoplasm of the muscle; it certainly offers an easy way out of the many difficulties which arise when we attempt to compare the twitch and the tetanus of the frog's gastrocnemius with the nicely adjusted movements of the living body, but it does so by postulating a mechanism of which we have as yet no direct proof and of a nature sufficiently vague to make disproof very difficult. For a detailed account of the evidence the reader must be referred to the papers of Bottazzi, Roaf [38], Langelaan [23], Dusser de Barenne [13], &c. The question will be referred to again in connexion with inhibition. For the present, it is justifiable to conclude that the impulses which govern the rapid contractions of striated muscle in the body are identical with those which can be reproduced experimentally in isolated nerve, but we must admit that there may be other kinds of nervous impulse of whose nature we are still quite ignorant.

CHAPTER II.—THE CHIEF CHARACTERISTICS OF THE IMPULSE IN PERIPHERAL NERVE.

§ 1.—*The All-or-None Principle.*

When the frog's sciatic nerve is stimulated mechanically, electrically or chemically, the gastrocnemius muscle contracts after a short but appreciable interval. The interval is longer when the stimulus takes effect at some distance from the muscle, and shorter when the distance is less. By measurements of this kind it can be shown that the stimulus sets up some disturbance in the nerve which travels down it at the rate of about 28 metres a second, and leads eventually to the contraction of the gastrocnemius. This disturbance is spoken of as the nervous impulse. Its nature is unknown, and the only direct accompaniment of it which can be detected with certainty is the electric response. The nerve in the region stimulated becomes electrically negative to other points on the nerve, and a "wave of negativity" spreads out in both directions with a velocity equal to that of the nervous impulse. There is possibly an evolution of CO_2 at the same time, but this is still rather doubtful [44].

Now, if two stimuli, separated by a short interval of time, are sent into the nerve, two nervous impulses are set up, two electric responses pass down the nerve and the muscle gives a summated contraction due to the fusion of two twitches. However, if the interval between the stimuli is reduced until it is of the order of 0·003 second, the second stimulus fails to set up any nervous impulse; the second electric response is absent and the muscle gives a single twitch only. In the same way if the stimuli are repeated with sufficient rapidity the nerve may fail to respond to every second or every third stimulus, and the number of nervous impulses may be only a fraction of the number of stimuli. This fact is of fundamental importance; it shows that the activity of the nerve-fibre cannot be continuous, and that conduction must always proceed by means of a series of discrete impulses which cannot follow one another at less than a certain interval. This interval is known as the refractory period; it varies with the temperature, the nature of the fluid surrounding the nerve, &c., but on the whole it is remarkably constant when the external conditions are not changed, and it is therefore extremely amenable to experimental analysis.

The mere existence of the refractory period leads to a very interesting conclusion as to the relation between the stimulus and the impulse [4]. It can be shown that the action of the external stimulus is to produce a local disturbance of a nature quite different from that of the nervous impulse; this local change takes a certain time to develop, and it must reach a certain intensity before the nervous impulse is set up at all; if it does not reach this intensity it subsides gradually when the stimulus ceases and nothing further takes place. Once the requisite intensity is reached, the nervous impulse is set in motion and the tissue enters into the refractory state. A second stimulus of the same strength occurring very soon after the first may increase the local change, but it will not be able to set up a second nervous impulse. Now if we reduce the time interval between the two stimuli to zero, the two acting together will have double the strength of the single stimulus; but, as before, the nervous impulse will be set up as soon as the local change reaches the required intensity and any further increase in its intensity will be without effect. So that, provided the stimulus is strong enough to set the impulse in motion, a further increase in its strength will not have any effect on the intensity of the impulse; in other words, the intensity of the impulse does not depend on the strength of the stimulus which sets it in motion. The process may be compared to that of firing a rifle by pressing the trigger. A certain force has to be applied to the trigger before anything happens, but the force may be increased indefinitely without causing the bullet to travel any faster.

It is, of course, well known that an increase in the strength of stimulus applied to a motor nerve trunk will lead to an increase in the force of contraction of the muscle, but here we are dealing with a structure composed of many nerve-fibres, and many muscle-fibres, and the increased contraction might be due simply to an increase in the number of muscle-fibres thrown into action. By experimenting with preparations containing very few nerve and muscle fibres Lucas [27] has shown that this is the case, and his results have been confirmed recently by Eisenberger [17]. If the muscle-fibre contracts at all it does so with a constant force which is independent of the strength of the stimulus. Thus there is an all-or-none relation between the strength of the stimulus and the contraction of the muscle-fibre. However, this gives us no information about the nervous impulse itself; we are dealing only with the beginning and end of a very complicated series of events, and it might be that the nervous impulse varied with the stimulus, even though the muscular contraction did not. Before considering this it will be necessary to arrive at some more definite understanding of what we mean when we speak of the intensity or size of the impulse.

From the point of view of the physics and chemistry of nervous conduction, the intensity of the impulse would mean the intensity of the change of state which spreads down the nerve-fibre. But we do not know what this change of state may be, and although it is accompanied by (and possibly identical with) the electric response, we do not know the exact relation between the two. Moreover, there are great experimental difficulties in measuring the electric response of a single nerve-fibre, and the response of a complete nerve-trunk will depend on the number of fibres in action as well as on the intensity of the change in each fibre. For this reason the measurement of the electric response in a nerve does not give much information as to the intensity of the impulse in each fibre, although such measurements certainly suggest that the intensity is independent of the strength of the stimulus [22]. However, from the present point of view, what really concerns us is not so much the intensity of the physical and chemical disturbance which is the basis of the nervous impulse, but the capacity of the impulse for propagating itself successfully down the nerve-fibre and overcoming any obstacles it may encounter in its passage through the central nervous system. In this sense it is legitimate to define the intensity of the impulse as meaning its capacity for conduction, and this capacity can be measured without much difficulty.

When a portion of the nerve-trunk is exposed to the action of a narcotic such as alcohol (or, indeed, to any set of conditions which leads to a depression of function without actually killing the fibres), it is found that a stimulus applied near the distal end of the narcotized tract will produce a contraction in the muscle, whereas one applied at a greater distance away will be unable to affect it, in fact there will be a limit to the length of narcotized fibre which can intervene between the point of stimulation and the muscle if the nervous impulse is to be conducted successfully [49], [20]. As the depth of narcosis is increased, the maximum distance through which the impulse can be conducted becomes smaller and smaller, and this holds good whether the stimulus takes effect within the narcotized region or on the normal nerve outside it on the far side from the muscle. As the whole of the narcotized tract is in the same state at any given stage in the narcosis, the facts can only be explained by supposing that the impulse becomes progressively less and less able to travel as it passes through the narcotized region. If it has only a short distance to go it will be able to pass the narcotized area and reach the normal fibre again, but if the distance is longer its ability to travel will be diminished to such an extent that the impulse will be extinguished completely. In other words, the intensity of the impulse suffers a progressive decrement as it passes through the affected region; when the depth of the narcosis is slight the decrement is small and the impulse can travel a long distance without extinction; when the depth is greater the decrement is more pronounced and the impulse is extinguished before it has gone very far. Evidently this furnishes a means of measuring the intensity of the impulse in terms of its ability to travel; we have only to arrange that the impulse whose intensity is to be measured shall pass through a region of progressive decrement, and the intensity will then be proportional to the distance which can be travelled in this area before complete extinction. This method is of great value in many ways, and it can be applied very simply to decide the question whether the intensity of the impulse varies with the strength of the stimulus or not. A muscle-nerve preparation is set up so that it can be excited by stimuli of variable strength at a point near the origin of the nerve. Between this point and the muscle is interposed a narcotizing chamber. The threshold stimulus (i.e., the stimulus which is just strong enough to give a muscular contraction) is first determined and the narcotic is then allowed to act gradually until the moment when this stimulus has just ceased to affect the muscle. At this moment the decrement suffered by the impulse in the narcotized area will be just great enough to extinguish it before it can pass out into the normal nerve beyond. However, an impulse of greater intensity should be able to pass through without extinction, and if a stronger stimulus is able to set up an impulse of greater intensity we should find that a stimulus two or three times as strong as the threshold would still be able to produce a contraction in the muscle. As a matter of fact, it is found that stronger stimuli have no such advantage; at the moment when conduction fails for impulses set up by a weak stimulus it fails also for those set up by stimuli many times as strong. This experiment [43], [25], [3] proves the conclusion already deduced from theoretical grounds, namely, that the strength of the stimulus does not affect the intensity of the nervous impulse which it sets up, provided only that the stimulus is strong enough to set up an impulse at all.

This shows that there is an all-or-none relation between the stimulus and the nervous impulse, but it does not show that the nerve-fibre cannot transmit impulses of different intensities if these can be sent into it by some means other than direct stimulation of the fibre itself, e.g., from the central nervous system. Evidently a narcotized fibre can transmit impulses of different intensities, though in this case the intensity does not remain constant as the impulse travels down the fibre; again, as will be seen later, the intensity of the impulse may vary with the state of recovery of the fibre. Thus the intensity is not absolutely invariable under all conditions, but this does not settle the question whether it is variable in the normal fibre. This point can be tested by much the same method as that described above. An impulse is made to pass through a narcotized region so that its intensity is reduced, and it is then allowed to emerge into the normal fibre beyond. Does its intensity remain smaller than it was at the beginning or does it recover to the normal value as soon as it has left the narcotized area? Its intensity is measured by determining its ability to pass a second narcotized tract and comparing it with that of an impulse which has been set up by stimulation at some point between the two narcotized tracts. This impulse will not have undergone any reduction in intensity when it reaches the second tract, so that its behaviour may be taken as an index of the normal. It is found that as great a depth of narcosis or as great a length of narcotized fibre is required to extinguish the impulse in this second tract whether it has been reduced by passage through the first tract or not. Thus the impulse must recover its normal intensity again when it passes out of the region of decrement into normal fibre, and it is therefore impossible to vary the intensity of the impulse in normal fibre by compelling it to pass through a region of imperfect conduction where the intensity will be reduced, the change in intensity is confined to the affected region and does not persist when the impulse emerges into normal fibre again [1].

These results are best expressed by the statement that in a normal nerve-fibre, where the impulse is not conducted with a decrement, the intensity of the impulse at any point depends only on the local condition of the fibre at that point and not at all on the previous history of the impulse before it arrives there. It does not depend either on the strength of the stimulus which set the impulse in motion or on the amount of reduction which the impulse may have suffered in regions of decrement as it passed down the nerve.

This is generally known as the "all-or-none" principle in nervous conduction. The foregoing is only a short abstract of the evidence on which it is based, but a more complete account may be found in the monograph on nervous conduction by Keith Lucas.

So far we have considered the impulse as an isolated event occurring in resting nerve, and in this case the intensity appears to be a fixed quantity, but in life it is unlikely that impulses ever occur singly except perhaps in the simplest of reflex actions. Consequently we have to consider how the impulse may be modified if it forms part of a series and passes through tissue which is still recovering from the effects of a previous impulse.

§ 2.—*The Recovery after the Passage of an Impulse.*

As we have seen, the immediate effect of the impulse is to make the nerve-fibre absolutely inexcitable and unable to conduct a second impulse. In the frog's sciatic at 15° C. the absolute refractory period lasts for about 0·003 second after the moment when the first impulse passes through the region under investigation. After this the excitability returns gradually. At first a very strong stimulus is needed to set up a second impulse and a weaker stimulus has no effect at all, even though it is several times as strong as the normal threshold, but as the interval between the two stimuli is increased the necessary strength of the second stimulus is gradually reduced, until at about 0·015 second it has fallen to the threshold value again. Following this comes a period of enhanced excitability in which the impulse can be set up by a stimulus weaker than the normal threshold for resting nerve [10]. In the frog's sciatic the increase of excitability is not very great and it is often difficult to detect at all, but there is no doubt that it exists, and in the nerve to the crayfish's claw the excitability may be increased to as much as 125 per cent. of the normal [33]. This period of increased excitability probably lasts until about 0·1 second after the passage of the impulse, though the end-point cannot be determined with much accuracy.

The changes of excitability are accompanied by parallel changes in the intensity of the impulse which can be set up during the different stages of recovery. During the absolute refractory period a second impulse cannot pass at all, even though it has been set up in some region where recovery is more advanced. During the stage in which the excitability is returning to normal the intensity of the second impulse is at first very small. As the interval between the first and second impulse is gradually increased by timing the second stimulus later and later, the intensity gradually rises until at about 0·015 second it is fully equal to that of the first impulse, which has, of course, the normal value for resting nerve. During the period of enhanced excitability the second impulse is greater than the first and this increase lasts for about the same length of time as the increased excitability. As before, the intensity of the second impulse is measured by determining the distance which it can travel in a region of decrement before it is completely extinguished and comparing this with the distance travelled by the first. When the second impulse is set up very soon after the first it is extinguished after a very short distance, but if it is set up during the period of increased excitability it will travel through a greater distance than the first.

Thus the whole course of recovery may be divided into three stages. There is first of all the absolute refractory period when all activity is abolished, then the period of depressed activity in which the nerve conducts an impulse of small intensity and needs a very strong current to excite it, and from this there is a gradual transition to the final period of enhanced activity when the excitability is greater than the normal and the intensity of the impulse also greater.

It is to be noted that for any given stage of recovery the intensity of the impulse which can be set up is still independent of the strength of the stimulus, provided this is great enough to set it up at all; also that the impulse is not conducted with a decrement although it may have an intensity less than the normal. As long as the impulse does not pass into regions where the state of recovery is altered it remains of the same intensity, but the intensity may be reduced by causing it to pass into a region at a lower temperature where recovery takes place more slowly, or increased by leading it into a region at a higher temperature where the recovery is more advanced.

The results so far described show that the intensity of a single nervous impulse, defined as its ability to pass a region of imperfect conduction, cannot be altered by changing the strength of the stimulus which sets it up, but that in a series of impulses the intensity of all but the first will depend on the state of recovery of the nerve and will, therefore, vary with the interval separating each impulse from its predecessor.

CHAPTER III.—SUMMATION AND INHIBITION OF IMPULSES IN PERIPHERAL NERVE.

These facts make it easy to see how a form of summation may be produced in peripheral nerve by the correct timing of a series of impulses. There is, however, another type of summation which must be considered briefly because it is apt to be confused with the true summation of impulses. We have seen that an external stimulus brings about a local disturbance in the nerve-fibre which must reach a certain intensity before the impulse is set up at all. A stimulus just below the threshold strength will produce a disturbance which is not quite intense enough to start the impulse. However, this local disturbance will take a short time to subside, and if another stimulus of the same strength is applied before it has subsided the additional disturbance may be great enough to reach the intensity required to start the nervous impulse. Thus two inadequate stimuli applied at the same point within a very short time interval may set up a nervous impulse where one alone would fail. This form of summation is purely local; it does not occur if the two stimuli are sent in at different points on the nerve, as the excitatory disturbance is confined to the point at which the stimulus is applied. The effect [28], [24] is generally known as the summation of inadequate stimuli, or as the summation of local excitation, and it is an entirely different process to the summation of impulses with which we are concerned at present.

The summation of impulses implies that a single impulse may fail to be conducted successfully, whereas two or more will succeed, and it is therefore concerned with the conduction of the impulses and not with their initiation. It may be brought about in peripheral nerve in the following way. If a muscle-nerve preparation is arranged so that a narcotic can be applied over a length of nerve between the point of stimulation and the muscle, it is possible to adjust the degree of narcosis so that a single impulse just fails to pass through the affected area. Thus a single stimulus, however strong, will not lead to any contraction in the muscle. But if two stimuli are sent in so that the second occurs about 0·03 second after the first, the second impulse will be set up during the phase of increased activity and its intensity will be greater than the normal. It will be able to withstand a greater decrement than the first impulse and therefore it will pass through the narcotized region and lead to a contraction. Further stimuli occurring at the right intervals may set up a train of impulses of which each occurs in the phase of heightened activity left by its predecessor, and in this way each succeeding impulse may be able to pass the region in which the first was extinguished [9]. It would be possible to extend this explanation to cover cases in which the contraction does not occur until several impulses have been set up and have failed to reach the muscle. Each impulse might travel a little farther through the region of decrement than its predecessor and each would facilitate the conduction of the impulse following it, so that in the end an impulse would succeed in passing through without extinction. However, in this case we are treading on uncertain ground as the conditions have not yet been reproduced in isolated nerve.

A form of inhibition may be produced experimentally by much the same mechanism. Suppose that the degree of decrement is not quite enough to extinguish a single impulse, and that impulses are sent in at intervals of more than 0·015 second. Each of these impulses will be of normal or supernormal intensity and therefore all of them will reach the muscle and a sustained tetanus will be produced. If the frequency of the impulses is increased to 0·007 second, either by stimulating more rapidly at the same point or by stimulating at another point as well, the impulses will follow one another so rapidly that each will occur during the period of depressed activity following the passage of its predecessor. Thus each impulse will have an intensity less than normal and will be unable to pass through the region of decrement; the muscle will relax because no impulses can reach it, and its contraction will remain inhibited until the frequency of stimulation is reduced again to its original value. This type of inhibition was first described by Wedensky [48], though its mechanism was not clearly understood until Lucas [30] had analysed it more thoroughly.

It is possible to produce the same kind of inhibition by increasing the strength of the stimuli without altering their frequency. In this case we must start with stimuli occurring at an interval as small as 0·007 second and of a strength only just greater than the threshold. The first stimulus will set up an impulse, but the second will occur during the period of depressed excitability and it will not be strong enough to set up a second impulse. The nerve will have recovered completely by the time the third stimulus reaches it, and this will set up an impulse of normal intensity. The fourth stimulus will fail as the second failed, but the fifth will succeed again and so on. Thus the number of impulses set up will be only half the number of stimuli in a given time, and the interval between succeeding impulses will be great enough to allow complete recovery to take place between each. However, a strong stimulus is able to set up an impulse during the period of depressed excitability, and so if the strength of the stimuli is increased every one of them will be able to set up an impulse instead of every other one. For this reason an increase in the strength of the stimuli will double the frequency with which impulses are sent down the nerve, and, as before, the intensity of each impulse will be reduced and the muscle will cease to contract. In this case increasing the strength of the stimuli is simply equivalent to increasing the frequency of effective stimulation and the mechanism of inhibition is unchanged [31], [2].

The foregoing account of peripheral inhibition and summation is somewhat at variance with that given by Verworn and Fröhlich, but their theory has already been discussed at some length by Lucas [35] and shown to be in conflict with the experimental evidence. In the particular case of the frog's sciatic there can be little doubt that the foregoing explanation is correct, and it is also correct for the nerve to the crayfish's claw. It has not yet been fully demonstrated in a mammalian preparation because the experimental difficulties are greater, but there is ample evidence to show that the course of recovery proceeds along the same lines and there is no reason to suppose that any new factors come into play.

The whole mechanism of peripheral summation and inhibition may be summed up in a very few words. If there is an obstacle to conduction in the course of the nerve-fibre this obstacle may be surmounted by timing a series of impulses, so that all but the first are greater than the normal, and it may become insuperable if the impulses are timed so that all but the first are smaller than the normal. In the former case each impulse must be set up during the phase of heightened activity left by its predecessor and in the latter case each impulse must be set up during the phase of depressed activity.

Before these results can be considered as anything more than a purely artificial effect we have to consider how far the experimental "region of decrement" produced by narcotics is at all comparable with any of the regions of imperfect conduction occurring naturally in the course of the intact nervous system.

CHAPTER IV.—NERVE-ENDINGS AND SYNAPSES AS REGIONS OF DECREMENT.

It is clear that conduction across the nerve-ending in a muscle-nerve preparation and across the synapse in the central nervous system differs in several respects from conduction in a simple nerve-fibre. It is much more easy to investigate the nerve-ending and therefore we will begin with this. Now in the first place it is evident that the nerve-ending, taken in its widest sense as implying the whole of the junctional area between typical nerve-fibre and typical muscle-fibre, is not simply a region of decrement and nothing more. It is a region where conduction proceeds in one direction only, where the local excitatory mechanism reacts to much briefer currents than usual, and where certain drugs have a peculiar effect. None of these features can be reproduced by establishing a region of decrement in a nerve-trunk. But there is fairly good evidence that the nerve-ending is often a region of decrement as well. When the preparation is perfectly fresh the nerve-ending may be able to transmit an impulse set up in the early stages of recovery and therefore of very small intensity, but a relatively slight change in the perfusing fluid, such as an addition of H' ions or absence of oxygen, will first of all increase the interval which must separate the two impulses, if the second is to reach the muscle, and will lead eventually to the failure of the first impulse also. A certain amount of confusion is introduced by the fact that we do not know how the rate of recovery of the nerve-ending is modified by these changes. Indeed the observation that in certain conditions the nerve-ending will no longer transmit a second impulse occurring very soon after the first does not show that there is any decrement in conduction. It might be due entirely to a slowing of the rate of recovery of the nerve-ending, the impulse being extinguished not because it is of small intensity but because it reaches the nerve-ending at a time when this is still in the absolute refractory state. However, the ultimate result of these changes is to make the nerve-ending unable to conduct even an impulse of normal intensity, and at this stage there can be no doubt that conduction with a decrement comes into play. The condition is best realized by fatiguing the preparation by repeated stimulation [7]. By this method it is easy to reach a stage in which a single impulse is unable to reach the muscle at all, whereas a series of impulses separated by about 0·03 second will produce a tetanic contraction. As we have seen, an impulse occurring 0·03 second after its predecessor will have an intensity greater than the normal because it occurs in the phase of increased activity. Thus the nerve-ending may be brought into a state in which it will refuse to conduct an impulse of normal intensity but will conduct one of intensity greater than normal; in other words, it will conduct with a decrement which is just great enough to extinguish an impulse of normal intensity.

In certain preparations, e.g., the crayfish's claw, this condition may develop spontaneously and remain unchanged for long periods [33]; in the frog's sciatic-gastrocnemius preparation it is only produced after repeated stimulation and it passes off very quickly, but it seems more than likely that it is always present in a certain percentage of the nerve-endings, even in a perfectly fresh preparation. Thus it is found that the electric response of the muscle in answer to the second of two stimuli in the nerve is considerably larger than the first response when the interval between the stimuli is about 0·03 second [39], [8]. The effect does not occur when the stimuli are applied directly to the muscle fibres and it is presumably due to the fact that a greater number of muscle fibres are thrown into action by the second stimulus because the impulses which it sets up are large enough to pass nerve-endings which will not transmit an impulse of the normal intensity. This effect is found in the sartorius as well as in the gastrocnemius, and it appears to be present even when the preparation, judged by other standards, is in a perfectly normal state. We have, therefore, a type of summation occurring at the nerve-ending and depending on exactly the same mechanism as the summation which occurs at a region of decrement artifically established in the course of a nerve-fibre. Indeed it seems probable that in life some of the fibres of each muscle can only be called into play by a summation of impulses at the nerve-ending [19].

Inhibition can be produced at the nerve-ending instead of summation by timing a series of impulses so that each is of smaller intensity than the normal [30]. In this case the preparation must be moderately fatigued, so that the decrement at the nerve-ending is not quite large enough to extinguish a normal impulse. It is then found that a series of impulses occurring at shorter intervals than 0·01 second will produce an initial twitch, corresponding to the first impulse, followed by complete relaxation, whereas a series with a frequency of 0·02 or 0·03 second will give a sustained tetanus. Here also the nerve-ending behaves exactly like a region of decrement produced experimentally in the course of a nerve.

We are treading on much less certain ground when we compare the synapse in the central nervous system with the nerve-ending and with an experimental region of decrement. The evidence is very scanty, but it is at least agreed that the chief differences between conduction in the central nervous system and conduction in a peripheral nerve are due to the peculiar properties of the synapse, or junctional region between one neurone and another. It is argued that the actual cell body and nucleus are probably concerned only with the nutrition of the fibres, since the conduction of the posterior root fibres is unaffected by the presence of the ganglion cells on them, and in certain invertebrates Bethe [14] has been able to destroy the whole of the nerve-cells completely without interfering with the reactions of the central nervous system. The fibres themselves probably conduct in much the same way inside the central nervous system as outside it, and so by a process of exclusion we can locate the characteristic reactions of the central nervous system at the synapse.

These conclusions need a good deal of qualification. It is often assumed that there must be some definite synaptic membrane separating one neurone from another, and to question this would be to stir up the smouldering ashes of the neurone controversy. But we have certainly no evidence that the properties ascribed to the synapse are confined to the exact point where one neurone leaves off and another begins, although the analogy with the nerve-ending lends a certain amount of support to this view. As a matter of fact, there is a large junctional area where the nerve-fibres are modified by splitting up into dendrites, network formation, &c., and the reactions attributed to the synapse may be due to the whole or to any part of this area. Indeed, the reactions of the synapse might be produced by any part of the neurone other than the axons which are histologically identical with peripheral nerve-fibres. However, it is clear enough that there are junctional regions in the central nervous system which behave very much like the nerve-ending, and whatever these regions may be histologically it is convenient to speak of them as synapses. They are susceptible to fatigue, to drugs and to lack of oxygen, they allow conduction in one direction only, they are often impassable to single impulses, and they admit of the summation and inhibition of a series of impulses. Whether this summation and inhibition is of the same nature as that produced at a nerve-ending or a region of decrement in simple nerve-fibre is a point which must be reserved for discussion until the next section, but although we have no rigid proof that the synapses are regions of decrement exactly comparable to those produced artificially, yet they are certainly regions of imperfect conduction, i.e., regions which only admit of the passage of an impulse under certain favourable conditions. Indeed, the whole working of the central nervous system depends upon this power of refusing certain impulses and allowing others to pass, and as the only form of imperfect conduction with which we are acquainted is that which occurs in a region of decrement, it is at least worth while to assume that the synapses are regions of decrement, and to see how this assumption tallies with the actual observations of conduction in the reflex arc.

There is at least one property in synaptic conduction which is absent in normal conditions both in peripheral nerve and in the nerve-ending as well. This is the property which enables a single impulse in the afferent nerve to set up a series of impulses in the efferent, so that the response may outlast the stimulus by several seconds. Veszi [47] has photographed the electric responses in the motor nerve of a frog under the influence of strychnine, and in this case a single stimulus applied to the posterior roots may set up ten or more impulses in the efferent nerve. This multiple response is an important factor in central conduction, but it does not necessarily imply any properties which are peculiar to the synapse, for a change in the ions of the perfusing fluid may easily bring a muscle or nerve into a condition in which a single stimulus will set up a large number of impulses. It is tempting to suppose that the effect depends on the increase of excitability which occurs in the third stage of recovery. If the initial excitability is very great this increase might be great enough to start a second impulse without any external stimulus. For instance, in the heart muscle the excitability rises to infinity after each refractory period is over and the tissue responds automatically; between this case and that of the motor nerve the difference appears to be one of degree only. However, it must be admitted that the rhythmic contractions of the frog's sartorius in a solution of NaCl may occur at intervals which are certainly too long to fit in with this hypothesis unless we assume that the time relations of the recovery process are considerably longer than usual [36]. Judging from Veszi's records the multiple responses in a reflex occur at intervals which would agree more or less with the periods of increased activity following each response. However, the precise mechanism of the effect is still very doubtful. Whatever this may be, the effect certainly exists and must be reckoned with in analysing the reactions of the reflex arc.

There is yet another point in which the reaction of the synapse differs from that of the nerve-trunk and that is in its great susceptibility to fatigue. The immense importance of fatigue in the economy of the central nervous system has been pointed out by Sherrington. As a rule a reflex path ceases to conduct at all after a short period of activity, whereas a peripheral nerve in the normal state will conduct without the least decrement after several hours' stimulation. The nerve-ending in muscle occupies an intermediate position, for the passage of a rapid succession of impulses through it increases the decrement in conduction and eventually leads to a complete block. The same fatigue effect is often met with in a nerve-fibre which has been narcotized so that it conducts with a decrement. By adjusting the strength of the narcotizing solution it is sometimes possible to arrive at a steady state short of complete failure of conduction, and in this state the decrement increases after the passage of a succession of impulses and returns slowly to its former value after stimulation has ceased. This fatigue is clearly of quite a different order to the momentary depression of function which constitutes the refractory state, and it is by no means certain that the refractory period is altered in any way when fatigue sets in. It is possible that the sole effect of fatigue is to increase any existing difficulty of conduction, though there may be a general depression of function as well. In any case when conduction occurs with a decrement, either in peripheral nerve or in the nerve-ending, the passage of a rapid succession of impulses does cause a temporary increase in the decrement, and presumably the same process may occur in the junctional regions of the central nervous system.

CHAPTER V.—CONDUCTION IN THE REFLEX ARC.

Taking all these differences into account we may construct the following model of the central nervous system. We may suppose it to consist of a number of neurones communicating with one another by junctional areas or synapses. The synaptic areas are regions of imperfect conduction; the impulse suffers a decrement as it passes through them, and this decrement is by no means constant, but varies, *inter alia*, with the amount of previous activity to which the neurone has been subjected. The duration of the refractory period and the extent of the change which occurs in the period of enhanced activity may vary in the same way and may have different values in different synapses or different parts of the same synapse. At some point in the synaptic region the passage of a single impulse may initiate a succession of impulses, and at some point conduction is possible in one direction only. The impulses which pass through the central nervous system are of the same nature as those set up in a simple nerve-fibre by an electric stimulus and they behave in the same way in regard to changes in intensity, passage through regions of imperfect conduction, &c.

This suggested scheme for the mechanism of the central nervous system has the advantage that it does not postulate any properties in central conduction which are not met with in a rudimentary state in conduction through simple tissues such as nerve or muscle fibre, and for this reason it should be possible to test it experimentally. It is true that there is very little direct experimental evidence at present, but we can see in a general way how the theory fits in with the observed facts of central conduction and where the chief difficulties are likely to arise.

In the first place we have to consider how the reaction of the central nervous system can be adjusted to different strengths of external stimulus if it is true that the size of the impulse follows the all-or-none principle in each nerve-fibre. Naturally a certain amount of grading is possible whenever there are a number of sensory end-organs of graded excitability, for in this case a stronger stimulus will set up impulses in a greater number of nerve-fibres. In the sensory organs of the skin it seems quite probable that the response of a single end-organ is of invariable intensity, for there is very little variation, if any, in the intensity of the sensation evoked by stimulating a single heat, cold, or touch spot. However, in the eye and the ear it is difficult to imagine that there are enough fibres to account for all the different intensities of sensation which can be perceived. The difficulty is not very serious, for the all-or-none principle is concerned with the relation between a single stimulus and a single impulse. If the stimulus sets up a train of impulses it is quite possible that their frequency, and therefore their intensity, might vary with the strength of stimulation. We have no direct proof that in the end organs of the eye and ear a momentary stimulus does give rise to a series of impulses, but there is evidence that a strong electric stimulus applied to a peripheral nerve-trunk may sometimes give rise to several impulses instead of one only, so that the suggestion has some experimental support. In any case it is clear that the effect on the central nervous system of a series of impulses will depend on the frequency with which the impulses recur, and as this frequency is variable there is no reason to assume that the excitation of a single end-organ must always have the same result, however the organ is stimulated.

From another point of view the all-or-none principle simplifies matters considerably. Whatever our conception of the central nervous system may be, there can be little doubt that the amount of energy leaving it by the efferent nerves may be considerably greater than the amount entering it as the result of an external stimulus. A light touch which cannot set up impulses in more than a few sensory nerve fibres may lead to the forcible contraction of a number of muscles, and in this case the number of motor nerve-fibres in action must be very much greater than the number of sensory fibres. It is scarcely conceivable that the loss of energy involved in the passage of impulses down the motor nerves should not be very much greater than the gain which results from the passage of a few impulses into the central nervous system. Evidently there must be some form of relay in the reflex arc to account for this multiplication of impulses. It would be possible to account for this by supposing that each neurone, or each nerve cell, may act as a relay, sending out impulses of full intensity whatever the intensity of the impulses reaching it from the afferent side. However, the all-or-none principle shows that this assumption is unnecessary, for any part of a simple nerve fibre may act in this way. We have seen that an impulse may be reduced almost to vanishing point by passage through a region of decrement, but that as soon as it re-enters the normal fibre again it regains its full intensity. This recovery could only take place if the energy involved in the passage of the impulse is supplied locally from each point in the fibre through which the impulse passes, just as the explosion wave in a train of gunpowder is maintained by the energy in each part of the gunpowder as the wave reaches it and sets it alight. Consequently there is no reason to suppose that there are definite relay stations in the central nervous system. Wherever an impulse passes down normal nerve-fibre its intensity will be the same whatever its former history may have been, and if an afferent fibre branches there is no reason to suppose that the impulses in each branch will be any smaller than the impulses in the original fibre in spite of the increased discharge of energy which will be necessary to maintain several impulses instead of one only. In the same way there is no reason to suppose that the impulse will become progressively smaller and smaller as it passes through successive junctional areas. It may be reduced in intensity in each area, but if it succeeds in passing through it will regain the normal intensity as soon as it leaves the region of decrement. Thus the relay effect of the central nervous system is simply due to the fact that it is a region where the afferent fibres branch repeatedly. Wherever this occurs there is the possibility of turning one impulse into two, and the increased energy required is supplied locally by the fibres themselves, just as it is supplied in peripheral nerve when an impulse regains its full intensity after reduction in a region of decrement.

If the central nervous system contains regions of decrement there should be no difficulty in accounting for at least one form of inhibition and summation. The presence of a region of decrement in peripheral nerve is the only condition necessary for the production of peripheral inhibition and summation and these effects depend simply on the normal course of recovery, according to which an impulse set up in the earlier stages of recovery has an intensity less than normal and an impulse set up in the later stages has an intensity greater than normal. If we consider the complications which are introduced in the central nervous system where the impulses have to pass through several relays of conductors and junctional regions with different rates of recovery, &c., it will be clear that the most varied possibilities are opened up without introducing any assumptions which have not been proved in the case of peripheral conduction.

The hypothesis is supported by various observations which show that the nature of the response in the reflex arc depends on the strength and frequency of the stimuli and also on the degree of decrement, just as it does in simple tissue. In the nerve-muscle preparation the inhibitory effect is produced by the extinction of impulses which follow so closely that they are all of subnormal intensity; thus the failure to reach the muscle will depend on the frequency of the impulses, the rate of recovery of the tissue and the degree of decrement which each impulse must sustain on its journey to the muscle. An increase in the strength of the stimuli may also lead to inhibition because it will increase the frequency of the impulses set up. Now in the case of reflex inhibition there are several instances in which an increase in the strength or frequency of stimulation converts an excitatory stimulus into an inhibitory. Sherrington and Sowton [41] found that stimulation of the popliteal by weak currents caused a contraction in the extensors, whereas strong currents gave inhibition. Tiedermann [45] investigated the effects of varying frequency of stimulation in the frog under the influence of strychnine. In this case an increase in the rate of stimulation changes the end-effect from excitation to inhibition, and there is a further likeness to peripheral inhibition in that a series of rapidly repeated stimuli sometimes produces an initial twitch followed by inhibition, just as it does in a fatigued muscle-nerve preparation. Again Tiedermann and Veszi [45], [46] have shown that inhibition may be produced by simultaneous stimulation of two roots in the cord when the stimulation of either root alone will give a contraction. Here also the effect would seem to depend on the increased frequency of the impulses which reach the final common path when both roots are stimulated at once. A reversal of the reflex effect is also produced by the action of certain drugs. Bayliss [11] has shown that stimulation which normally leads to a vaso-constrictor reflex gives vaso-dilatation instead after chloroform has been administered. Sherrington and Sowton [42] found the same reversal in a limb reflex. The most important action of chloroform in a nerve-trunk is to produce a decrement in conduction and therefore it is only to be expected that in the central nervous system it should increase the already existing decrements and so lead to the extinction of impulses which were formally just large enough to reach the motor neurone.

If we grant that the synaptic regions may have different rates of recovery there is no difficulty in explaining the fact that stimulation of an afferent nerve may lead to contraction of one muscle and inhibition of its antagonist. We must suppose that the frequency of the impulses reaching the final common path of the inhibited muscle is so great that all the impulses are of subnormal intensity and are therefore extinguished before they reach the muscle. The frequency of the impulses reaching the muscle which contracts must be slower, so that the impulses shall be large enough to pass. These conditions will be realized if at some point on the path leading to the muscle which contracts there is a region which recovers so slowly that every other impulse from the afferent nerve arrives during the absolute refractory period and is extinguished completely. The effect of this will be to halve the number of impulses passing towards the muscle, so that each will be large enough to avoid extinction; in the other path, where the impulses do not pass through a region of slow recovery, the frequency will be great enough to cause inhibition.

Most of the phenomena of reciprocal innervation may be accounted for by a mechanism of this type. However, difficulties begin to arise when we come to consider the balanced effects of inhibitory and excitatory stimuli. Sherrington [40] has shown in the case of voluntary muscle, and Bayliss [12] in the vaso-motor reflexes, that if an excitatory and an inhibitory nerve are excited at the same time the effect produced is a simple algebraic summation of the two single effects and depends entirely on the relative strengths of the two stimuli. Now, the frequency of the impulses in the central paths should be, if anything, greater when both nerves are stimulated than when the inhibitory nerve is acting by itself, and therefore the impulses should be still further reduced in intensity. Indeed, the stimulation of several afferent nerves in connection with the final common path should always result in inhibition, whatever their effects might be when acting alone. Veszi and Tiedermann have shown that this does occur in the spinal frog under the influence of strychnine, but Sherrington's records prove that the result is by no means constant. A strong stimulation of an excitatory nerve may break through a simultaneous inhibition and cause a contraction, and in certain cases rhythmic contractions are set up by stimulating an excitatory and an inhibitory nerve at the same time. To explain these effects we should have to introduce various subsidiary hypotheses as to the excitability of different nerve-fibres, the changes introduced by fatigue, &c., and though the suggested scheme is sufficiently elastic to admit of elaboration along these lines, it would be a mistake to insist on this until the evidence is much more complete. For the present we must be content to point out that the difficulty exists.

One of the most important features of the scheme suggested to account for reflex conduction is that the neurones are supposed to behave like peripheral nerve-fibres in regard to the impulses passing through them. We have assumed that the activity of the neurone cannot be continuous, but that each impulse must be followed by a short period of inactivity which passes off gradually in the various stages of recovery. As Forbes and Gregg have shown, when the motor nerve is excited reflexly the impulses which pass down it are indistinguishable from those set up by direct stimulation, and we have no direct proof that any other form of activity is possible either in the nerve-fibre or in the neurones of the central nervous system. However, the reflexes which have been investigated from this point of view have all been of the type resulting in rapid contractions of the muscle and we know very little of the tonic postural reflexes. Buytendick [16] has shown that the electric response of a muscle in decerebrate rigidity has the usual oscillating character, but, as we have seen, there is a certain amount of evidence in support of the view that the tone of a muscle is controlled by a mechanism entirely different from that which regulates its rapid phasic contractions. The evidence is too complicated and, as yet, too inconclusive to be discussed in detail, but it shows that we must face the possibility that there may be some way of producing a continuous activity in certain neurones and certain parts of the muscle, an activity quite unlike the intermittent discharges which form the usual type of nervous impulse. If this is so, the production and inhibition of tone cannot be explained by the mechanism we have suggested, but all inhibition is not necessarily of the same nature and there is no reason to suppose that the inhibition which cuts short an active contraction is not identical with that produced experimentally in a region of decrement or nerve-ending. In this case the contraction is certainly maintained by a series of impulses in the motor nerve and its inhibition is due to the extinction of these impulses. Indeed, it is rather difficult to imagine that the tonic contraction in voluntary muscle is not produced and inhibited by the same mechanism, for when a contracting muscle is inhibited it relaxes completely and it seems an unnecessary complication to suppose that the relaxation is produced by two distinct mechanisms, one cutting short the phasic contraction and the other the residue of tone.

It would be idle to continue these speculations, for they have already gone far beyond the existing experimental basis. The suggested mechanism of central conduction may well prove to be completely absent or present only in a small degree in the central nervous system. However, the hypothesis has the advantage that it does not demand any properties in central conduction which cannot be shown to exist in some degree in simple conducting tissues, and as all these properties can be analysed without much difficulty in simple tissues it should be a fairly straightforward task to determine how far they will account for central conduction. The hypothesis may have been set up only to be knocked down in the near future, but it is at least tangible enough to knock down and the process cannot fail to reveal something of the mechanism of the central nervous system.

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STIMULATION OF THE MOTOR CORTEX IN A MONKEY SUBJECT TO EPILEPTIFORM SEIZURES.

BY C. S. SHERRINGTON, M.D., F.R.S.

IN the higher mammals epileptiform convulsions can, as Magnan showed, be evoked by injection of absinthe; that strong faradization of the motor cortex may in the dog and monkey evoke an access of spasm of epileptoid character is a well-known laboratory experience. Stimulation of the cortex in an animal suffering from idiopathic epilepsy is, however, a laboratory experience of rarer occurrence; I have not succeeded in finding an instance of it recorded. The following observation may therefore appear worth mention here.

A "Jew" monkey (*Macacus fuliginosus*) received into the laboratory this spring was found to be subject to epileptoid attacks. These attacks had a characteristic form. The opening movement visible was retraction of the left angle of the mouth, whence contraction spread to all the facial muscles of that side and to the eyes. Then followed spasm of the neck, and of the left arm, and then of the left leg. The animal fell over to the left side. Finally, the right side became involved, though not so violently as the left. The attack lasted usually about two minutes, the animal lying seemingly unconscious, though with blinking eyes. The tongue was not bitten, nor was urine or fæces passed. When the convulsions had subsided the animal lay panting and would gaze about helplessly as though wondering what had happened. For a short period after each attack the limbs appeared to be paretic.

One cannot tell how often the animal was taken by these seizures; six seizures were, however, observed in the course of one week. In every observed instance the attack seemed to be induced by the taking into the mouth of a large morsel of food; indeed, it was found that the attack could be in some measure expected after giving the animal a too bulky slice of apple. The animal appeared in quite good health except for the epilepsy; it was an adult male.

After the animal had been under observation for some weeks, the motor cortex of both hemispheres was exposed under full chloroform narcosis. The cranial vault was found to be abnormally thick; where trephined in the upper parietal region the bone thickness measured 6 mm., which is about twice the usual measurement. The exposed cortex was tested by faradism with the unipolar method.

Abnormality of reaction was discovered nowhere except in the "tongue" area of the right hemisphere clearly, and less clearly in the "tongue" area of the left hemisphere. The threshold value of stimulus of the "tongue" area did not seem unusually low, or lower than that of such areas as "thumb or index finger," but each stimulation of the area unless extremely brief, e.g., less than 1 sec. and sometimes even then, provoked a tongue movement which almost immediately became clonic and epileptoid, meaning by the latter that it continued as a series of discharges after the faradic stimulus of the cortex had been withdrawn, and although it usually did not spread, it required a considerable period, e.g., a minute, for its subsidence. If the stimulus were continued for a few seconds the epileptoid movement did spread from the tongue to the muscles of the left angle of the mouth; occasionally it would involve all the facial muscles of the left side. By no persistence of the faradization could, however, the epileptoid movement be made to extend beyond the face. It never, unlike the natural seizures observed in the animal, spread to the neck or limbs even of the same side, let alone of the opposite.

The cortical area for retraction of angle of mouth itself did not seem to give abnormal responses; the area where opening of the jaw could be obtained seemed somewhat more extensive than usual, but otherwise normal. In the left hemisphere the tongue area yielded "epilepsy" discharges easily, though not so readily as did the tongue area of the right cortex, nor did the epileptiform discharge spread. Elsewhere throughout the motor region the reactions appeared entirely normal.

No gross indications of an anatomical kind were visible suggestive of abnormality in the cortex of either hemisphere.

In evaluating the experimental results it is to be remembered that limited epileptiform discharge is in the monkey's cortex usually obtainable by prolonged or quickly repeated faradic stimulations of almost any point in the motor cortex. The result observed with the tongue area of this animal was therefore a quantitative rather than a qualitative one. On the other hand, the exceptional readiness with which tongue "epilepsy" could be evoked is remarkable because, at least in my experience, the tongue area is not one from which epilepsy is usually easily provocable. Comparing the experimental result with the seizures observed in the animal clinically, it had not been noted for the latter that they began in the tongue. They may have done so and that point have escaped observation owing to the tongue lying hidden in the mouth.

INTRACRANIAL ANEURYSMS.

BY HUBERT M. TURNBULL, D.M.

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My colleague, Dr. Fearnsides [1], in an article upon "Intracranial Aneurysms," published recently in this Journal, has honoured me by references to my paper [2] on "Alterations in Arterial Structure, and their Relation to Syphilis." Dr. Fearnsides, on pages 236 to 238, has attempted, by analysis of the statistics scattered throughout my paper, to show the relative frequency with which aneurysms within the cranium and within the body had been found in the Pathological Institute of the London Hospital. The result has demonstrated to me many defects in my writing. In the hope of removing these defects, as far as possible, I have obtained permission from Dr. Fearnsides to revise the analysis.

A comparative analysis of all the lesions tabulated in my paper was rendered impossible by my having in places analysed the records for 1908 to 1913, inclusive, and in places the records for 1907 to 1913, and by my having omitted the total number of examinations in the years 1908 to 1913. In order to draw up a comparative table it is necessary either to add to some of my statistics missing numbers for 1907, for instance twenty-six cases of syphilitic aortitis, or to deduct from others the numbers belonging to 1907. The latter form of correction gives the most accurate figures, because during the year 1907 our methods of controlling examinations and records were in process of evolution. When this correction is chosen, it is obvious that the total number of examinations during the years 1908 to 1913 must be supplied. Both Dr. Fearnsides and I have adopted as a definition of true aneurysm all dilatations of the whole arterial wall. I shall, therefore, first draw up a table, on similar lines to that of Dr. Fearnsides, so as to include all aneurysms which fell within this definition. I shall then, however, discuss the disadvantages attendant upon a comparative table in which this definition is employed, and the criticisms which I have received in respect of this definition. Finally, I shall attempt to remove these disadvantages, and meet these criticisms, by a second table based upon a modified definition of "aneurysm."

During the years 1908 to 1913, inclusive, in a total of 6,829 necropsies, there were 6,751 examinations of the body, and 4,547 examinations of the head; in 78 of these examinations of the head, examination of the body was not permitted.

TABLE I.

True Aneurysms of Aorta :—	
Due to syphilitic inflammation	175 cases
,, tuberculous inflammation, 1 example in	1 case
,, non-granulomatous inflammations, 4 examples in	4 cases
,, medial degeneration (focal, 18 cases ; diffuse, 4 cases +) ..	22 + cases
	202 + cases
Extensive Dissecting Aneurysms of Aorta :—	
Due to medial degeneration	5 cases
,, developmental deficiency	2 ,,
	7 cases
True Aneurysms of Other Large Elastic Arteries :—	
Due to syphilitic inflammation, 42 examples (all in cases included in above 175 cases of syphilitic aortitis) in	29 cases
,, medial degeneration (diffuse aneurysm)	+ ,,
	29 + cases
True Aneurysms of Small Elastic Arteries :—	
Due to tuberculous inflammation, 10 examples (all aneurysms in pulmonary vomicæ; many such aneurysms had doubtless escaped detection) in	10 cases
True Aneurysms of Muscular Arteries (excluding the Cerebral Arteries) :—	
Due to syphilitic inflammation, 6 examples (all in cases included in above 175 cases of syphilitic aortitis) in	5 cases
,, infective embolism, 16 examples in	14 ,,
,, spread of non-granulomatous inflammation from without, 3 examples in	3 ,,
,, medial degeneration (focal, saccular, 12 examples in 7 cases; focal associated with atheroma + ; diffuse +)	7 ++ cases
	29 ++ cases
True Aneurysms of Cerebral Arteries :—	
Due to infective embolism, 15 examples in	13 cases
,, medial degeneration following congenital developmental deficiency, 38 examples in	29 ,,
,, ,, ,, (diffuse + ; focal associated with atheroma +)	++
	42 ++ cases

This revised list gives, I believe, a true comparative analysis of our records. It is, however, of no service for exact comparison, because I am unable to give under every specified cause either the exact number of examples of aneurysm or the exact number of cases in which aneurysms occurred. To estimate the total number of aneurysms, as defined by Dr. Fearnsides and by me, would, for instance, in most cases of syphilitic aortitis, have been an almost impossible task owing to their great number. Still greater difficulty would have attended an attempt to ascertain and record the exact number of cases exhibiting certain forms of aneurysmal dilatation of which I merely mentioned in my paper the frequent occurrence. Thus, in regard to aneurysmal dilatation due to medial degeneration of the aorta, I recorded eighteen cases in which there were "small pitted or wrinkled areas," and four cases of "very marked, diffuse dilatation." In a preceding paragraph, however, I mentioned that diffuse dilatation of the aorta, especially of the thoracic aorta, was a very frequent sequence of medial degeneration and "is easily recognized in almost every male over the age of 50." I also stated that in the other large elastic arteries, "slight diffuse dilatation frequently results from medial degeneration." Again, in regard to aneurysmal dilatation of the muscular arteries due to medial degeneration, I recorded, in Table VII, seven cases in which there were saccular dilatations of muscular arteries other than the cerebral. I had, however, previously mentioned (pp. 244-5) that diffuse dilatation due to medial degeneration "is frequently found in the muscular arteries," including the cerebral arteries, and that "slight focal dilatation may be present at the sites of atheromatous plaques," this being "almost constant in atheroma of the cerebral arteries."

Even if I was in a position to make the above table complete by the substitution of exact numbers of cases for the sign +, the list would, as I have learned from correspondence, give a false impression to the majority of readers. This is the consequence of the definition of "true aneurysm" which has been adopted by Dr. Fearnsides and by me. I believe that our definition, which includes every dilatation of the whole wall, is both the only scientific and the only really practicable definition. This definition includes, however, a very large number of dilatations which the clinician, at any rate, would never regard as aneurysms. Indeed, one kindly critic wrote that he regretted my "old-fashioned" definition, and would have had me apply the term aneurysm to saccular dilatations alone. In a general discussion on the causation of aneurysm it would obviously be impossible to accept no dilatation as an aneurysm unless it was saccular. The small crateriform pits and narrow sulci, for instance, which are almost constant in syphilitic aortitis, are all potential saccular aneurysms. Areas which show, post mortem, only such minute dilatations may be more widely dilated during life; thus, the aortic commissure may post mortem only show small sulci in an intima conspicuously thickened by inflammation, and the aortic valves may be competent to the water test, although clinical observation and the post-mortem appearances of the heart prove that relative incompetence of the valves had been present during life. There are diffuse dilatations which all investigators would call aneurysms, and which give clinical signs and symptoms similar to those caused by saccular aneurysms. Further, I have found it impossible to estimate exactly what is to be regarded as a saccular dilatation. For exact definition of a saccular dilatation some arbitrary proportion between depth and diameter would require to be determined and universally accepted.

I realize, however, the desirability of attempting to distinguish between what might be called "practical" and "academic" aneurysms; I realized this when preparing my paper. Without such a distinction it would appear impossible to compare our figures with those of other observers. The aneurysms in the cerebral arteries tabulated as due to congenital weakness and to infective embolism, and certain aneurysms in the other muscular arteries due to medial degeneration, were unquestionably saccular and would be regarded by all as aneurysms. In the cases, however, of syphilitic inflammation of the aorta, other large elastic arteries and muscular arteries, and in the cases of medial degeneration of the aorta, I was unable to determine a satisfactory distinction between the practical and academic dilatations. I attempted to make a distinction by quoting the number of dilatations which had given rise to clinical manifestations by rupture, by pressure, or, in the case of aortitis, by causing relative incompetence of the aortic valves. A distinction based on such clinical criteria is, however, obviously imperfect. In a fresh attempt to make the distinction, I have made the following modification in our definition. I have only accepted as aneurysms: (1) definitely saccular dilatations; (2) saucer-like dilatations; and (3) sharply defined, focal, fusiform dilatations. For convenience I have referred to the shallow, saucer-like dilatations as "pouches." With this modified definition I have compiled Table II. This table includes, I think, all the dilatations which would have been accepted as aneurysms by all. In the case of syphilitic inflammation of the aorta and other large elastic arteries, and especially in the case of medial degeneration of the aorta, dilatations are also included which would probably not be considered, by the majority, worthy of the term aneurysm. Inasmuch, however, as Dr. Fearnsides compiled his table in order to show the exceptionally large number of cerebral aneurysms in the records of the London Hospital Pathological Institute, it is well that, if the modified definition should err, it should err by including too many aneurysms of arteries other than the cerebral.

I regret that I am unable even with this modified definition to give the exact number of aneurysms in syphilitic aortitis; in several cases small examples have merely been recorded as "multiple."

TABLE II.

Focal, Saccular, Saucer-shaped and Fusiform Aneurysms, 1908-1918—*Aorta*:—		
Due to syphilitic inflammation (in 28 cases shallow pouches only)	Examples, 114 +	in 83 cases.
,, tuberculous inflammation	,, 1	,, 1 ,,
,, non-granulomatous inflammation	,, 4	,, 4 ,,
,, medial degeneration (all very small or shallow pouches)	,, 4	,, 4 ,,
	Examples, 123 +	in 92 cases
Other Large Elastic Arteries:—		
Due to syphilitic inflammation (all cases are present also in the above 83 cases of syphilitic aneurysms of aorta)	Examples, 6	in 6 cases
Small Elastic Arteries:—		
Due to tuberculous inflammation (in vomicæ) ..	Examples, 10	in 10 cases
Muscular Arteries (excluding Cerebral Arteries):—		
Due to syphilitic inflammation (1 case is present also in the above 83 cases of syphilitic aneurysms of aorta)	Examples, 2	in 2 cases
,, infective embolism	,, 16	,, 14 ,,
,, spread of non-granulomatous inflammation from without	,, 3	,, 3 ,,
,, medial degeneration	,, 12	,, 7 ,,
	Examples, 33	in 26 cases
Cerebral Arteries:—		
Due to infective embolism	Examples, 15	in 13 cases
,, medial degeneration following congenital developmental deficiency	,, 33	,, 29 ,,
	Examples, 48	in 42 cases

In regard to aneurysms caused by syphilitic inflammation of the large elastic arteries other than the aorta, and of the muscular arteries, it must be emphasized that in all the cases recorded in this table and in Table I the syphilitic inflammation involved vessels close to the aorta, and the aorta was affected by similar inflammation. Thus, of the syphilitic aneurysms of the large elastic arteries recorded in both tables the aneurysm farthest from the inflamed aorta lay in the upper two inches of the right common carotid, the lower inch of this artery also showing evidence of inflammation to the naked eye; of the syphilitic aneurysms of the muscular arteries the aneurysm farthest from the inflamed aorta lay in the centre of the splenic artery. This aneurysm is one of the two recorded in Table II. It was associated with gummata in the peritoneal tissue round the pancreas, in addition to aortitis. In this case the infection had probably arisen independently of the aortitis. In all the remaining cases of syphilitic inflammation of the large elastic and muscular arteries in Table I and Table II the inflammation would appear to have been merely an extension from the aorta.

If Table II is accepted for purposes of comparison, then aneurysms of the aorta occurred in 92 cases or in 1·36 per cent. of examinations of the body. Aneurysms of large elastic arteries other than the aorta occurred in 6 cases; all these aneurysms were due to syphilitic inflammation and were accompanied by aneurysms of the aorta. Aneurysms of muscular arteries, other than the cerebral arteries, occurred in 26 cases, or 0·38 per cent. of examinations of the body; only 2 of these aneurysms were due to syphilitic inflammation; both of these were accompanied by syphilitic aortitis, 1 by an aneurysm of the aorta. Aneurysms of cerebral arteries occurred in 42 cases, or in 0·92 per cent. of examinations of the head; in 13 cases the aneurysms were caused by infective embolism, and [illegible] of these 13 cases are recorded also in the list of cases of embolic aneurysms in other muscular arteries.

If aneurysms in tuberculous vomicæ and in peptic erosions or ulcers are omitted, the following table compares the number of cases in which aneurysms were found in the arteries of the body with the number of cases in which aneurysms were found in the head, during the years 1908 to 1913:—

Cases with aneurysms of arteries in the body .. 117, *i.e.*, 1·78 per cent. of examinations.
" " " " *cranium* .. 42, " 0·92 " " "

The cases which Dr. Fearnsides quotes, from the literature, of aneurysms of cerebral arteries caused by syphilitic inflammation, remind me of another, serious, omission from my original paper. I should have stated explicitly that I had attempted to give an account of personal observations on alterations in arterial structure, that I had given references to papers to which I was indebted for information, but that I had not examined the literature in order to ascertain to what extent my observations and conclusions corresponded to those of others. My observations were not confined to the cases given in my tables. At the time the paper was written I had seen over 9,000 necropsies in two general hospitals. It was perhaps presumptuous to decide what conditions were rare on this material alone, although, on the other hand, rare cases are more likely than common to be recorded in the literature. In this connection I may add that a subsequent 1,500 necropsies have not given me an aneurysm due to syphilitic inflammation in a cerebral artery. Immediately after publication I regretted, for another reason, this omission of an explicit statement of the method in which my paper was written. I realized that in its absence I would appear to claim priority for conclusions which other workers had already published. I had, and have, no doubt that the great majority of my observations and conclusions, if correct, could be found in previous publications; there must be very many, besides those quoted by Dr. Fearnsides, whose work I had merely confirmed.

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BRAIN.

PART II, VOL. 41.

SENSATION AND THE CEREBRAL CORTEX.

BY HENRY HEAD, M.D., F.R.S.

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CHAPTER I.—INTRODUCTION.

WE tend habitually to speak of all varieties of general somatic sensibility in terms of touch, pain, heat and cold; for these categories seem to accord best with what we gather from introspection. To these elementary sensations it is customary to add a hypothetical "joint-sense" and the whole complex is supposed to be transformed by association and judgment into the mental processes by which we become aware of the physical qualities in the world around.

Moreover, according to the doctrine of specific nerve energy, as it is usually held, each psychical act of sensation is associated on the physical side with certain distinct processes, which, starting in the peripheral end-organs, pass unaltered to the cortex of the brain. Here, according to the usual supposition, each specific form of nerve energy evokes those changes which underlie its peculiar aspect of sensation.

Such a view of the physical mechanism and nature of sensory processes was not unreasonable in an age ignorant of the gradual evolution of the human nervous sytem. Man was thought to have been created perfect, armed with apparatus to receive and conduct the special processes, which underlie each fundamental sensory experience revealed to him by introspection.

This conception of man as a created being has long passed away, but the manner of regarding sensation, to which it gave rise, still dominates psychological teaching. Even the neurologist, who is in daily touch with the consequences of lesions to the nervous system, tended, until recently, to speak of sensory impulses as if they were invariably grouped into those subserving touch, pain, heat and cold. This arose in the main from two causes. His almost complete preoccupation with structure rather than function compelled him to consider the central nervous system as something stable, developed it is true from that of lowlier organisms, but fixed and unalterable in its activities. He did not recognize that all the processes of its functional evolution were still evident in its physiological activities. Even now, the conception that a stimulus may be adequate or entirely ineffective, according to the conditions which have preceded its application, has made little impression on practical neurology.

Preoccupation with the sensory effects produced by lesions of the spinal cord also inclined the clinician to accept the fundamental division of afferent impulses into those for touch, pain, heat and cold. For spinal injuries notoriously tend to disturb these four modes of sensation independently of one another. But, as soon as disorders of sensation came to be studied more closely, it was obvious that something further was required to explain even so simple a problem as the changes produced by a lesion at this anatomical level of the nervous system. Four specific groups, corresponding to touch, pain, heat and cold, were obviously insufficient to contain all the impulses originated at the periphery by the impact of a stimulus. For it is now universally acknowledged by those who have had the opportunity of observing cases of injury to the spinal cord, that all these forms of sensibility may be preserved, and yet the patient be unable to recognize the spacial relationships of the affected limb, or to appreciate movements carried out passively. Power to recognize the posture of the affected limb, to discriminate the two compass-points applied simultaneously and to appreciate the vibration of a tuning-fork may be disturbed, although every other form of sensory activity is perfect.

As soon as it was clear that these aspects of sensation depended on the integrity of the posterior columns, it was thought imperative that they should receive a name. Nothing could have been more unfortunate for the progress of knowledge than the names selected. Clinicians, seeking for a sign that would reveal the condition of the posterior columns, concentrated their attention on one or other of the groups of impulses passing upwards in this portion of the spinal cord, oblivious of the general principles underlying their conduction. Hence the terms, "joint-sense," "deep sensibility," "vibratory sense," and "stereognosis," which give a most inadequate view of the part played in sensation by the posterior columns.

For the neurologist, who speaks of the "joint-sense" as dependent on these columns, forgets that pain is a most important element of the sensations arising from joints. "Deep sensibility" is a sufficiently accurate name for those impulses which arise in subcutaneous structures, such as bones, joints and tendons; but it can never be disturbed as a single group, except by a lesion of the deep afferent fibres of the peripheral nerve. The changes, which result from destruction of the posterior columns of the spinal cord, correspond to a fraction only of such impulses.

On the other hand, "bathyæsthesia" is, strictly speaking, of too special a significance. It was invented to indicate the faculty of recognizing the position of the limbs in space and of appreciating the vibrations of a tuning-fork, in as far as they were disturbed by lesions of the spinal cord. But an injury of the posterior columns may also destroy the power of appreciating the size, shape and relative weight of objects placed in the affected hand; to this loss of sensibility the inadequate name of "astereognosis" has been applied. For "stereognosis" signifies ability to recognize the solidity of objects by touch, and cannot by any stretch of terminology be made to include the power of appreciating weight.

Any attempt to fit the sensory disturbances, produced by injury of a nerve trunk, into four primary categories of touch, pain, heat and cold, tends to even worse confusion. For, when one of the larger nerves is divided, it may be that pressure can be perfectly appreciated, though sensibility to lighter contacts is abolished; and pressure may evoke pain in parts insensitive to a prick or a burn. In the same way, discrimination of two points may be destroyed, though recognition of posture and passive movement is maintained unimpaired. Moreover, as far as the peripheral mechanism is concerned, sensibility to heat can be shown to be a complex process, involving the simultaneous activity of several different end-organs.

As each new fact of sensory dissociation became established, the conception of the part played by the nervous system became more confused and the nomenclature more chaotic. The psychologist, who was not in direct contact with the facts, was content to explain the phenomena which underlie sensation as divisible into four groups of specific impulses aided by a hypothetical "joint-sense"; these, elaborated by the mental processes of association and judgment, seemed all that was required. The attitude of the neurologist varied between an uncritical agnosticism and a determination to square his observations on injuries of peripheral nerves with his conception of spinal dissociation.

Obviously, some link was missing in the chain of reasoning which would harmonize those forms of sensory loss presented by lesions of the spinal cord with the apparently impossible inductions of those who worked on the peripheral nervous system. This was found in the conception that afferent impulses underwent regrouping on their way from the peripheral end-organs to their final termination in the highest receptive centres. Here was the means by which peripheral impulses, with their opportunist or developmental grouping, could be recombined in accordance with their sensory qualities.

Each peripheral end-organ is a specific resonator attuned to some aspect of physical vibration. It responds by producing an afferent impulse stamped with the characteristics peculiar to the organ in which it has arisen. Each of these impulses acts in turn on a series of receptive mechanisms within the central nervous system, which are themselves attuned to certain physiological qualities; here, those of like sensory disposition are gathered together, whatever their peripheral origin. Peripheral end-organs respond to physical changes in their environment, whilst these endo-medullary receptors react specifially to the welter of afferent impulses which reaches the central nervous system.

In this way, afferent impulses of like sensory quality become gathered together in their passage through the spinal cord and brain-stem. At the termination of the fillet, their grouping so closely corresponds to the four qualitative aspects of sensation, with the addition of certain spacial attributes, that it would almost seem as if no further integration were necessary.

But it is in the subsequent fate of these regrouped afferent impulses that the bankruptcy of the older conceptions of sensory activity is most evident. It has long been recognized, that a gross lesion of the so-called "sensory cortex" does not abolish any of the four primary qualities of sensation. An injury of the spinal cord or brain-stem may produce complete loss of sensibility to pain, heat or cold in the affected parts; any one mode of sensation may be destroyed or preserved independently of the others. But when we attempt to investigate the effects of a lesion of the cerebral cortex on sensation, the problem seems insoluble; the patient appears to be untrustworthy, sometimes he appreciates a touch, at others a stronger stimulus passes unrecognized. Heat and cold are not confused, but the patient may be uncertain whether a warm object is hotter or colder than one of an obviously different temperature, which he had held in his hand a few minutes before. Inconsequent answers, hallucinations, uncertainty dominate the records from the affected parts; he cannot be said either to appreciate or not to appreciate tactile or thermal stimuli; and yet he may be remarkably trustworthy to every test applied elsewhere.

The clue to this condition, so disconcerting to the views of the introspective psychologist, is given by the behaviour of the optic thalamus. Until recently, the part played by this organ in sensation was unknown. But we now recognize that it is the seat of those physiological processes which underlie crude sensations of contact, pain, heat and cold, together with the feeling-tone they evoke. The essential organ of the optic thalamus is the centre for the affective aspect of sensation, whilst discrimination and spacial projection are the product of cortical activity.

Thus it is evident that, between the response of any peripheral end-organ to a physical stimulus and the final impact of the afferent impulse on the sensory centres of the cortex and optic thalamus, lie the levels of physiological activity. At the one pole is the external universe, whilst at the other we reach the primary mental states of sensation and feeling. Between them the diverse effects, produced by a physical stimulus on the receptive mechanisms of the body, are subjected to infinite modifications of which we can never be conscious under normal conditions.

This conception of levels of activity within the nervous system was one of the leading aspects of Hughlings Jackson's teaching. To him a "level" was always one of function rather than of structure; when he spoke of the "cerebellar level," he implied the peculiar influence which he attributed to the action of the cerebellum on the motor apparatus. But the overwhelming preoccupation of neurologists with anatomy has led them to adopt the convenient term "level" in an anatomical rather than in a physiological significance. Throughout this paper, I shall use the word in its strictly functional sense, unless it is preceded by the word "anatomical." For the activities of any one "anatomical level" may be widely different in the functional hierarchy. Injury to the sensory cortex not only disturbs the power of attention and discrimination but also produces hypotonia. An even more remarkable example is seen when the spinal cord is gravely injured at any one point, so as to destroy its lateral and posterior columns; the anatomical level is given in terms of segments, but the consequences from the point of view of afferent impulses belong to at least two functional levels. Destruction of the posterior columns has blocked peripheral impulses running in their primary combinations; the lesion of the lateral columns, on the other hand, has interfered with impulses of the secondary level, which have already undergone qualitative regrouping.

It is obvious, therefore, that, if we are to understand the sensory effects produced by a lesion of any part of the nervous system, we must record the reaction of the affected parts to each test in turn, noting which stimulus is capable of evoking its appropriate sensation and which of them is not appreciated, or induces an abnormal response. All general nomenclature must be avoided and we must be careful not to speak in terms applicable to one sensory level, when describing the changes which occur at another. Then only can we say that, at this particular level, sensory impulses are grouped in such and such a manner.

This has been our aim throughout the series of researches on disturbances of sensation in which I have collaborated during the last fifteen years. A number of selected tests were systematically applied in cases of definite injury to various parts of the nervous system, between the periphery and the cerebral cortex and the results interpreted in physiological terms.

The object of the present paper is to demonstrate, by the same method of dissociated sensibility, the manner in which the cortex cerebri modifies the afferent materials it receives, to produce those physiological states which so closely underlie sensation.

But, in order that the nature of this activity may be clear, it will be necessary to recapitulate the various changes to which afferent impulses are subjected at different levels of the nervous system. None of these could be deduced by *a priori* reasoning. If I take into my hand a glass containing hot water, I receive the impression of its size, shape, weight, temperature, roughness or smoothness; I also know that it causes me pleasure or discomfort according to the heat of the water it contains. Each of these qualities, recognizable in my impressions of the glass of water, have behind them many sensory impulses, which have been modified and regrouped between their peripheral origin and central termination.

The actual afferent consequences produced by the glass of hot water cannot be deduced from any *a priori* reasons. All we can discover from introspection is that the object is of a certain size, shape, temperature and consistence; we must for ever remain ignorant of the nature of the impulses which underlie these sensory characters. But by observing the consequences produced by interference at various known points in the nervous system, we find that some impulses are intercepted, whilst others pass on to reach the highest receptive centres and form the underlying basis of an abnormal sensation. By analysis of such dissociations of sensibility, due to lesions at different levels of the nervous system, it is alone possible to unravel that vast mass of mechanical dispositions, which lie normally outside the field of consciousness.

CHAPTER II.—THE INTEGRATION OF AFFERENT IMPULSES.

§ 1.—*The Peripheral Afferent System.*

Let us first of all consider the principles which emerge from the application of this method to the peripheral nervous system.

If the whole skin and superficial structures are rendered insensitive over an area of considerable size, without at the same time disturbing any of the nerve fibres running from deeper parts, sensation is not abolished, but is profoundly modified. The affected part still retains many sensory faculties usually attributed to end-organs in the skin; and yet it is innervated solely by the deep afferent system supplying the connective tissues, muscles, joints, and tendons. The experiment on my arm showed that pressure and jarring contacts were quickly appreciated, and localized with remarkable accuracy. Roughness was recognized as perfectly as on the normal hand. Even contacts, which would usually have been called "light touches," evoked a sensation provided they depressed the surface of the skin; but grave deformations caused by pulling the hairs passed entirely unnoticed. A large part of our normal sensibility to touch depends on this deep system of afferent fibres, which was allowed to remain intact in my experiment ([13] p. 355).

To these deep end-organs we also owe the power of responding to certain aspects of pain. For, although the denervated area on the back of my forearm and hand was completely insensitive to the pain of a prick and a burn, the discomfort produced by excessive pressure was even more pronounced than over similar parts on the normal hand. Moreover, the cramp caused by repeated electrical stimulation of the muscles was at once appreciated as a most unpleasant sensation. Evidently, then, pain as we know it in daily life may be the result of two sets of impulses, one starting from the surface and the other from the end-organs of the deep afferent system.

We have already mentioned the accuracy with which the position of the spot touched can be localized, when a part is endowed with deep sensibility only. Another faculty, which depends entirely on the integrity of this afferent system, is the power of recognizing movement, and appreciating the position of any part of the limb. Here, impulses from the surface of the body play little or no part; in all cases where this aspect of sensibility was found to be disturbed from a lesion of peripheral nerves, we were able to prove, that the deep afferent fibres had been injured in some way by the lesion. Yet in spite of the ease with which the spot touched can be localized and posture appreciated, the two points of the compasses cannot be discriminated, when they are applied strictly simultaneously. Here we discover the first of those dissociations in spacial recognition, so disconcerting to the conventional psychologist.

Evidently, the deep afferent system is adapted to react to all contact stimuli, other than the lightest touch or movement of the hairs. It is also the means by which we become aware of the situation of a stimulated point and of the posture and movement of any part of the body or limbs. Again, it is the channel for evoking all those forms of pain and discomfort, caused by undue pressure, injury to the joints or abnormal muscular contraction.

So far the dissociation of function can be produced with ease and observed without difficulty. But, when we attempt to analyse the functions of the afferent mechanism which innervates the surface of the body, the problem is less easy experimentally. We are obliged in most cases to fall back on the process of regeneration to produce that dissociated sensibility, which reveals the composite nature of the cutaneous afferent system.

In the course of the experiment on my arm, we found that sensibility began to return to the skin a little over six weeks after the operation; within seven months the back of the hand was sensitive to pain, to cold, to heat and to any contact which moved the hairs. But the sensation experienced was peculiar and quite unlike any reaction from normal parts; for, although less easily evoked, it was unusually vivid. The pain of a prick was intolerable; cold was said to be colder and a suitable warm stimulus produced a more actively pleasant effect than over the normal skin. In this stage of recovery a high threshold was associated with a brisker response ([13] p. 369).

The sensation aroused within the affected area had certain other peculiar characteristics. It radiated widely and was not confined to the neighbourhood of the stimulated spot. Portions of the affected area seemed to be linked up together, so that stimulation of the one evoked a sensation of the same specific quality referred to the other. Thus, brushing the hairs, pricking or the application of heat or cold to the neighbourhood of the index-knuckle caused a diffuse outburst of the appropriate sensation over the dorsal aspect of the thumb. Moreover, the response was massive, diffuse and bore little relation to the measured strength of the stimulus, so long as it was effective ([13] p. 412).

It had long been known that certain minute spots in the normal skin were especially sensitive to heat, to cold, and to pain, although there was reason to believe they were not the cause of all its sensory functions. We were able to show that this punctate system was responsible for the peculiar mode of reaction, present during the first stage of recovery. This was particularly easy in the case of thermal stimulation. For, whenever a part of the affected area became sensitive to heat or to cold, one or more of these specific spots was discovered within it. Thus, five months after the operation, we found that a certain part of my thumb had become sensitive to cold; whenever the silver test-tube containing iced water was brought into contact with the dorsal surface of the first phalanx, it caused a brisk, diffuse sensation of cold. On testing this part with an ice-cold rod of not more than 1 mm. in section, a single spot was discovered, to which the sensibility of the whole area was due. No part of the affected skin on the back of the hand reacted to heat until nearly six and a half months after the operation. Then, for the first time, a region in the neighbourhood of the head of the first metacarpal bone responded to the warm test-tube. Here a minute spot was found, which subsequently proved to be one of the most constant and active of all the heat-spots on my hand.

These minute areas of specific sensibility can be discovered in the normal skin by suitable methods of examination; but they are not the only organs responsible for its sensibility, as is the case during the first stage of recovery. The skin between them is sensitive to touch and to temperature. This makes it impossible to carry out on the normal hand, many of the most illuminating observations made, in my case, during the period when the skin was endowed solely with this punctate mechanism ([13] p. 429).

For sensibility to contact at this stage was due solely to the hairs; if they were carefully shaved away, I no longer responded to cotton wool moved lightly over the surface. Heat and cold could only be appreciated in parts, where we were able to demonstrate the existence of minute areas with their appropriate specific reaction. If, as was not infrequently the case, a portion of the skin, one or more centimetres square, was devoid of heat-spots, that part did not respond to temperatures between 40° and 45° C., even if stimulation was made with a flat-bottomed tube covering the whole of that area.

These heat- and cold-spots are scattered irregularly about the surface of the body. Within an area of 5 cm. square upon the back of my hand, we discovered sixteen heat-spots during the first stage of recovery. These were not increased in number as sensation became further restored; on the contrary, some of them became more difficult to discover, owing to the way in which their vivid reaction was controlled. The cold-spots within the same area in my case numbered about sixty-eight in all, of which twenty-eight belonged to the first grade in constancy and vividness of reaction; they were rarely, if ever, missed in the records extending over more than four years. For it must be remembered that one of the most striking characteristics of organs belonging to this punctate system, even in the normal skin, is the profound influence of general fatigue on their reaction. After a tiring day I could mark out those of first grade only, and could obtain no reaction from many second-grade spots, which were easily discovered after a night's rest.

The pain produced by a prick at various points within the affected area varied greatly in severity and in the ease with which it could be evoked. But the pain-spots are so numerous within any one centimetre square that their position cannot be determined with the certainty of those for heat and cold.

But all the organs of this punctate mechanism possess certain characteristics in common. They regenerate with great rapidity after the peripheral nerve has been successfully united, and for many months the reaction of the skin to contact, to pain, to heat and to cold shows that they alone are responsible for any sensibility it may possess.

Each sensory spot reacts to adequate stimuli in a strictly specific manner. Thus, when a cold-spot is stimulated by a suitable metal rod recently removed from iced water, the sensation is one of cold; but when the same rod, heated to between 45° and 50° C., is brought into contact with a cold-spot, the sensation evoked is equally one of cold. Ice and water at 45° C. happen to be adequate to excite the cold-spots, and in both cases the result on consciousness is the same, in spite of the physical difference in the stimulus.

Another important and characteristic function of this punctate system of cutaneous sensibility is the mode of its reaction. This can be tested most accurately by experiments on the heat- and cold-spots. The response to a stimulus capable of exciting one of these organs is not strictly graduated by its intensity, but is arranged on the "all or nothing principle." This reaction occurs over a limited range, the heat-spots responding to temperatures above about 38° C., the cold-spots to those below about 26° C. Once the stimulus is effective, it matters little how cold or how hot it may be; end-organs of this class indicate its quality, not its intensity.

A natural consequence of this mode of reaction is the overwhelming importance of the extent of surface covered by the stimulus rather than its intensity. For it is obvious that if one cold-spot can be excited to vigorous activity by 20° C., a stimulus at the same temperature, sufficiently extensive to cover many cold-spots, will seem to be far colder although in reality its intensity is the same. Thus, whenever we are dealing with these primitive end-organs, extensity is of greater sensory importance than intensity. This is exquisitely shown by the following experiment, carried out repeatedly on my hand, during the period when it was innervated solely by this punctate system of end-organs. A group of cold-spots was carefully marked out and one of them was stimulated with an ice-cold rod which just covered it; the sensation produced was one of cold. But, when a small flat-bottomed tube containing water at 20° C., was placed over the whole group of spots, I thought it was much colder than the iced rod. Time after time the more extended stimulus of less intensity produced a greater sensory effect than the more intense stimulus of less extent ([13] p. 422).

I have already pointed out, that one peculiarity of the reaction from any part of the surface, innervated solely by this punctate system, is the diffuseness and wide radiation of the sensation evoked. When any hair-clad part, such as the back of the hand, is stroked with cotton wool an intense "tingling" and "itching" is produced over widely remote parts of the affected area. This sensation disappears, when the skin is carefully shaved, and is due to movement of the shaft of the hairs; it is not a function of the skin as a whole. In the same way, any effective hot, cold or painful stimulus produces it sspecific sensation, which is referred to the same remote parts of the affected area, irrespective of any difference in quality.

This punctate afferent mechanism we have called "protopathic," on account of its primitive characteristics. These are shown in the early restoration to activity of its end-organs after the nerve has been reunited. Secondly, each set of end-organs responds in a strictly specific manner, as shown by the remarkable phenomenon of "paradox cold"; for it matters little to the cold-spots whether the stimulus is at 15° C., or 45° C., provided it is capable of evoking a response. If so, the sensation is one of cold. A third primitive character appears in the nature of the reaction. The end-organs of this system tend to react on the "all or nothing" principle, and stimulation evokes a response, not strictly graduated according to its intensity. This explains the predominant effect of the extent of the stimulus and the relatively small part played by its intensity. Lastly, all sensations, which arise from uncontrolled protopathic activity, tend to radiate widely and to be referred into remote parts, a condition incompatible with a high degree of spacial discrimination.

Superposed on this older mechanism is another cutaneous system of later development and higher functions, which we have called "epicritic." When a nerve trunk has been successfully united, heat-, cold- and pain-spots are restored to full activity, many months before there are any signs of returning epicritic sensibility. But, as soon as it begins to appear, the mode of reaction changes. The diffused sensations, so characteristic of protopathic activity, give place to a more strictly localized response; radiation no longer occurs into remote parts of the affected area. At the same time, the power returns of distinguishing two compass-points, applied simultaneously. This is essentially an ability to recognize objects in two-dimensional space, and it is this faculty which checks and controls the diffuse reaction, characteristic of protopathic sensibility. For, if we are conscious of the extent of surface covered by the stimulus, radiation into remote parts and wide diffusion become impossible.

The "all or nothing" reaction of the heat- or cold-spots also gives place to one graduated more closely according to the intensity of the stimulus. At the same time, the affected part becomes sensitive to temperatures that lie in the middle of the scale (27° to 38° C.).

Another remarkable change, due to the restoration of epicritic sensibility, is shown in the return of the power of adaptation to varying temperatures. If, under normal conditions, the hand has previously been exposed for some time to warmth, an object at a certain temperature, such as 29° C., may seem to be "cool," whilst conversely, if it has been adapted to cold, the same stimulus may appear to be "warm." So long as my hand remained in the protopathic condition, no such adaptation was possible, and any adequate thermal stimulus was invariably thought to be hot or cold, irrespective of the temperature to which it had been previously exposed ([13] p. 406).

The activity of the epicritic system is essentially modulated according to the intensity and locality of the stimulus. It is concerned with the finer degrees of tactile and thermal discrimination and in this way is opposed to, and controls, the diffuse "all or nothing" reaction of protopathic sensibility.

It has been objected, that protopathic and epicritic sensibility are not the outcome of the activity of two peripheral systems, but are stages in the restoration of function of the divided nerve. In fact, many observers call the former a state of "paræsthesia," and are satisfied that they have thereby sufficiently explained its peculiar reactions. The improbability of such a view is shown by the following observations made on my hand.

Throughout the protopathic stage of recovery, the vividness and extent of the reaction became steadily greater with the gradual return of sensibility to pain and the increasing number of heat- and cold-spots. But this massive and radiating response was curtailed, or even abolished, at the height of its development with the first signs of returning sensibility to light touch and to minor degrees of heat. Had protopathic sensibility been but a half-way house on the road to recovery, we should have expected its steady increase to be associated with a simultaneous decrease in radiation and reference. But this is not the case; the return of epicritic impulses diminishes protopathic activity, as expressed in the sensations evoked by adequate stimulation of the end-organs of this system.

How completely this is due to control by a separate and dominant system over the activity of one of more primitive functions was shown by the behaviour of my hand when cooled. Epicritic sensibility is liable to be affected by the general action of external cold, especially before it has been completely restored. At a time when almost the whole of the back of my hand had so far recovered that referred sensations could no longer be produced, it was rapidly cooled; radiation and reference returned as vividly as of old, and the hand was thrown back for a time into a purely protopathic condition. The newly recovered activity of the high-grade epicritic mechanism was disturbed by the cold, and protopathic impulses, previously inhibited, now passed on uncontrolled.

This control can be exerted, even by epicritic impulses from the adjacent normal skin, if normal and abnormal parts are stimulated simultaneously. When a cold tube was placed so that it fell just within that part of the dorsum of the hand which was in a purely protopathic condition, a vivid referred sensation was always experienced in the thumb. But when the base of the tube fell partly within this area and partly over the adjacent normal skin, reference to the thumb was abolished. The only sensation produced was one of coldness around the spot on the back of the hand in contact with the tube.

Occasionally it happens by a fortunate chance that, after division of cutaneous nerves, some part of the denervated area retains its epicritic sensibility, though insensitive to prick and to heat and cold. With the return of these aspects of sensation, pain-, heat- and cold-spots make their appearance, but are accompanied by none of the usual radiation and reference into remote parts. For in this case protopathic sensibility is restored to a part already actively epicritic; it therefore comes back under control from its first restoration.

Some might object that such dissociations are purely pathological, the product of extraordinary conditions, in no way comparable to normal sensory states. But in the glans penis we find an organ endowed with sensibility to deep pressure, but otherwise reacting exactly in the protopathic manner. It is insensitive to light contacts, insufficient to arouse sensations of pressure-touch. It is endowed with pain-spots and with heat- and cold-spots which react vividly to appropriate temperatures only. The response is diffuse, and the extent of the stimulus is of more importance than its intensity. Here, then, is a normal organ endowed with an exactly similar sensory mechanism to that which we believe exists as a primitive afferent system in the skin ([13] p. 388).

Such a form of sensibility is capable of an affective, rather than a discriminative, reply to external stimuli. It forms an admirable warning mechanism; for it can give either a painful or a pleasurable answer, which admits of no hesitation. Repulsion or attraction must follow as soon as a sensation of this order is developed.

With the advent of the epicritic mechanism, the response is no longer massive and diffuse, but strictly localized. This renders it easy of control, and admits of choice in the motor response. The surface of the body can also become adapted to stimuli to which it has been exposed for any length of time; thus, the same external physical condition no longer evokes of necessity the same sensation, and the central nervous system ceases to react with the same vigour to constant stimulation.

Here, in the dominance of a reaction based on intensity over one dependent mainly on the extent of the stimulus, we see the elements of that struggle between the discriminative and affective aspects of sensation, which forms so important a factor in the activity of the human mind.

§ 2.—*Synthesis and Control of Afferent Impulses underlying Sensations of Pain, Heat and Cold.*

All the incongruous impulses, generated in the various peripheral end-organs, can never affect consciousness at the same moment; some succeed in forming the basis of sensation, whilst others are inhibited and would never be recognized were it not for the facts of dissociated sensibility. During their passage through the central nervous system they sooner or later undergo integration carried out, partly by combination into specific groups and partly by selective inhibition. Of these two processes the former, as far as painful and thermal sensibility are concerned, takes place at the first synaptic junction in the spinal cord.

As soon as the impulses arising in the deep system and in the two cutaneous mechanisms reach the spinal cord, they undergo regrouping. All those capable of evoking pain, whether from the surface or from deeper structures, are picked up by intramedullary receptors, which guard the entrance to the secondary tracts, devoted to conduction of this specific aspect of sensibility. In the same way, the afferent consequences of all stimuli capable of evoking a sensation of heat, whether of epicritic or protopathic origin, are gathered together into special secondary tracts. An analogous synthesis occurs of those impulses, arising from stimuli on the cold half of the thermal scale.

The underlying factors of these three primary modes of sensation are selected and integrated, to pass on in separate tracts as high as the optic thalamus. As soon as the first synaptic junction is passed, on the passage upwards of afferent impulses, the characteristic features of protopathic and epicritic sensibility disappear. A lesion of the spinal cord, which destroys sensibility to pain, tends to abolish it, whatever may be the source of the impulses on which it depends. In the same way with heat or with cold, each specific aspect of sensation is disturbed, regardless of the system of end-organs in which the physical changes arose. Moreover, sensibility to pain, to heat or to cold may be affected independently of one another, a condition impossible as a consequence of any lesion of the peripheral nervous system. We believe that these intramedullary receptors may be compared to resonators placed in a concert hall; they pick up, and respond to, those impulses only to which they are attuned, irrespective of the instrument that produced them. ([8] p. 642).

The changes, which occur at the first synaptic junction, result in the sorting of afferent impulses into specific sensory groups; those originated by similar properties of an external stimulus are gathered together, although they may have arisen in end-organs of different peripheral systems.

But such specific combination would in the end be useless without selective inhibition. A temperature of 45° C. can stimulate the heat-, the cold- and the pain-spots, and gross confusion would result, if such a stimulus were able to evoke at the same moment sensations of pleasant warmth, ice-cold and pain.

The following experiment shows how complete may be this selective inhibition. Take a metal rod with a blunt end measuring not more than 1 mm. in diameter and cool it to 15° C. or below. On carefully exploring the back of the hand, spots will be discovered where the rod produces a peculiarly vivid sensation of cold. These are the cold-spots. Now heat the metal rod to 45° C. and place the end so that it exactly covers one of the spots; if the experiment is carefully carried out, you will experience a sensation of coldness, as if the rod had been dipped in cold water. This is the condition known as "paradox cold." Take a flat-bottomed. metal tube, containing water at 45° C., of such a size that it will cover many cold spots and the skin around them. Lay this on the back of the hand and you will experience a pure sensation of heat; and yet we know that this temperature is capable of evoking a sensation of cold, if it is applied to each of the cold spots individually. Under normal conditions, however, the effect produced by stimulating the cold-spots is dominated, before it can disturb consciousness, by the coincident impulses, due to the action of the same external physical agent on the mechanism which responds to heat.

The behaviour of the glans penis forms another excellent illustration of such inhibition. It is not uncommon to find that the punctate end-organs with which it is endowed are not uniformly distributed; the tip of the glans around the meatus may be devoid of heat-spots, but sensitive to cold and to pain. In such a case we carried out the following remarkable experiment. The end of the penis was dipped into a glass containing water at 40° C.; since no heat-spots were present and this temperature has no effect upon the cold-spots, the only sensation evoked was a peculiarly disagreeable pain. When, however, the temperature of the water was raised to 45° C. pain was to a great extent displaced by a vivid sensation of cold, due to stimulation of the cold-spots. An elevation of temperature, which might have been expected to evoke greater discomfort, ceased to be strictly painful, because of the appearance of the specific sensation of cold. But around the corona the penis is always well furnished with heat-spots, in addition to those for cold and for pain; as soon, then, as the water at 45° C. covered the corona, without reaching the foreskin, both cold and pain disappeared, giving place to an exquisitely pleasant sensation of heat ([13 p. 390].

The following observation made on my hand, during the later stages of regeneration, shows the same phenomena from another aspect. The dorsal portion of my thumb had so far recovered that contact with a large vessel containing water at 40° C. produced a pleasant, well-localized sensation of warmth. But, at the same time, the skin in the neighbourhood of the index knuckle was in a condition of pure protopathic sensibility, and a cold tube applied to this area evoked a diffuse and vivid outburst of cold referred to the back of the thumb. It was, therefore, possible to stimulate the skin of the thumb with heat, and, simultaneously, to evoke a sensation of cold referred to the same area. As soon as this was brought about, all sensations of heat and of cold ceased in the thumb and were replaced by pain. When, however, the cold tube was removed from the neighbourhood of the index knuckle, the direct effect of the hot stimulus in contact with the thumb reasserted itself, as pleasant warmth and the pain disappeared.

In none of these instances was the process of selective inhibition in any way conscious. The sensation evoked was a definite one of heat, of cold or of pain. The whole process takes place on the physiological level and would have remained for ever incomprehensible, had it not been for the phenomena of sensory dissociation.

Unlike the regrouping of afferent impulses, this selective inhibition does not occur at the first synaptic junction. For, in cases where an intramedullary lesion destroys all sensibility to heat without affecting that to cold, a temperature of 45° C. applied to the anæsthetic parts may still evoke paradox cold (Head and Thompson [8] Case 7, p. 678). Moreover, it is possible to show that, under converse conditions, temperatures below 22° C. may produce an analogous paradoxical sensation of heat from regions entirely insensitive to cold. All observers have found difficulty in proving the existence of this phenomenon by experiments on the normal skin. But, when all sensibility to cold is lost owing to a lesion of the spinal cord, and yet the patient remains sensitive to heat, temperatures below about 20° C. may excite sensations of warmth ([8] Case 8, p. 682). These observations are particularly easy to carry out in suitable cases of lesions within the spinal cord, because of the large areas which may not respond to one form of thermal stimulus, although they are acutely sensitive to the other.

In the same way, if all sensibility to heat and cold is abolished in consequence of an intramedullary lesion, but the appreciation of painful stimuli is unaffected, it is easy to show that temperatures between 40° C. and 45° C. are capable of producing pain. Thus, a single physical stimulus, such as a temperature of 45° C., may start changes in peripheral end-organs, which pass up all the secondary tracts devoted specifically to the impulses of pain, of heat and of cold. Final integration by selective inhibition must take place at a level above the termination of these columns in the optic thalamus.

§ 3.—*Tactile Sensibility and Localization.*

Painful and thermal stimuli generate impulses that are more easily analysed by the dissociation method than those due to contact of some external object with the surface of the body. But tactile sensibility is equally the result of incompatible impulses which never, under normal circumstances, affect consciousness, until they have undergone combination and selective inhibition.

When any considerable area of the skin is rendered insensitive without disturbance of deep sensibility, as was the case in the experiment on my hand, that part of the body still responds to contact. Slight pressure on the abnormal area can be appreciated and localized, whilst touches with cotton wool and deformations of the surface, caused by pulling the hairs, remain completely unperceived. Whether a sensation was or was not elicited, when a thick camel-hair brush was applied to the dorsum of my hand, depended largely on the way the brush was used. If applied suddenly and vertically to the skin, so as to cause a jar, a slight tactile sensation was produced; but, when the pressure was made more gradually, it was not appreciated until the brush was distinctly bent. In the same way, stroking the part gently with a wisp of long-fibred cotton wool was entirely unperceived; but cotton wool balled together into a "swab," such as is used for sponging a wound, caused a sensation, if pressed upon the affected part. The more gradually contact was established and the smaller the pressure applied, the less likely was it that a sensation would result. All observations show, that parts endowed with deep sensibility are especially sensitive to jarring impact.

Measurements revealed that, whereas the normal skin on the back of my hand was sensitive to the contact of hairs of from 8 to 12 grm./mm.^{2}, the part innervated by the deep system alone responded to 35 grm./mm.^{2} but not to 23 grm./mm.^{2}.

Deep sensibility may, therefore, endow the body with a remarkable power of responding to contacts so slight that, under ordinary conditions, they would have been spoken of as "light touches." This is no pathological state; for, during the period when the skin of my hand was entirely insensitive, I frequently found extreme difficulty in marking out the affected area by means of light pressure. When the back of my hand was stimulated by pressure with the round head of a pin, no essential difference could be noticed between the quality of the sensations on the normal and affected parts.

This power of recognizing contact carries with it remarkably accurate localization. When, before any recovery had taken place, I was asked to indicate the spot touched, my answers were as accurate on the one hand as on the other.

This faculty of indicating the situation of a touch was not associated with any power to discriminate two points applied simultaneously to the skin, even when separated to the greatest distance possible over the affected area on the back of my hand. But two points applied successively were at once recognized, even when they were not more than 1·5 cm. distant from one another. Here then we find a remarkable dissociation between the one- and the two-dimensional aspects of spacial sensibility. A single spot, or two spots touched in succession, could be localized with normal accuracy, but the strictly simultaneous stimulation at two points on the surface of the body could not be recognized as more than one contact.

With the return of protopathic sensibility, this power of localization is profoundly disturbed. For one of the chief characteristics of the reaction of this primitive cutaneous system is its diffuseness and tendency to cause sensations referred to remote parts. If, during this stage of recovery, cotton wool was rubbed over a small hair-clad portion of the affected area a peculiar widespread tingling was produced. This response had the same fundamental features as that from the heat-, cold-, and pain-spots. It was strictly confined to the hairs and was absent from hairless parts, such as the radial aspect of the thenar eminence. If all the hairs were carefully shaved, the protopathic area became insensitive to cotton wool and all diffuse reaction to contact ceased. Moreover, the remote sensation due to stimulating the hairs with cotton wool was referred into exactly the same parts as the pain evoked by pulling them individually, or by exciting the heat- and cold-spots of the same area.

So long as the affected portion of my hand was innervated by deep sensibility only, the position of a touch was well localized, if sufficiently heavy to be appreciated. With the first signs of returning protopathic sensibility, localization became gravely disturbed, for there are but few stimuli of daily life which do not excite some other sensation beside those of contact. The more nearly the stimulus consisted of pure tactile pressure, the easier was it for me to indicate the spot to which it was applied. But, when it was produced by rubbing the hair-clad parts firmly with a swab of cotton wool, or by pressure with the end of a cold test-tube, the diffuse radiation was so great that no correct localization was possible. Here the return of sensibility was actually associated with disturbance of a power already possessed by the affected parts.

With the first return of epicritic sensibility, reference occurs less frequently and radiation is diminished. The affected parts become sensitive to touch, even when the hairs are shaved, and measured tactile stimuli of 21 grm./mm.^{2} or less are distinctly appreciated. During this stage of recovery it grows increasingly possible to recognize the duality of two spots touched at the same moment, and the compass-test begins to show the existence of some tactile discrimination ([13] p. 411).

All tactile impulses, whatever their peripheral origin, become ultimately combined by the time they reach the upward termination of the spinal cord. But the course they follow differs from that of impulses arising from thermal and painful stimuli, in that a double path is open to the tactile impressions of each half of the body throughout the greater part of their intramedullary course. In the larger number of cases of loss of sensation produced by a lesion in the opposite half of the spinal cord, pain, heat and cold only are affected. But, in a few instances, this loss is accompanied by a definite disturbance of tactile sensibility; such change is always associated with some injury to the posterior columns on the side of the loss of sensation, accompanied by more or less destruction of the antero-lateral aspect of the opposite half of the spinal cord. For, in order that tactile sensibility may be affected from an intramedullary lesion, it is necessary not only to injure the crossed secondary paths, but also to disturb the conduction of the primary path for tactile impulses in the posterior columns. For this reason, sensations of touch and pressure are much less frequently affected in cases of Brown-Séquard paralysis than those of pain, heat and cold.

If, however, tactile sensibility is either abolished, or even diminished, it is affected as a whole. Impulses, originating from the surface and from the deep parts, have been combined and all distinction is lost between those arising in different peripheral systems. The lightest touch and the heaviest pressure, short of discomfort, form the two ends of a graduated tactile scale. Whenever it would seem as if some lesion of the spinal cord had destroyed sensation to light touch, but left that to pressure unaffected, careful measurements will show that the tactile loss is greater than could be accounted for by the abolition of cutaneous sensibility alone. Not only is light touch abolished, but the lesser grades of pressure are also affected, and many stimuli, which would have been adequate to excite deep sensibility, fail to arouse appreciation.

It is probable that, in this synthesis of tactile impulses, the peculiar effect produced by brushing the hairs of my hand in the protopathic stage still remains as the quality we recognize as "tingling" or "itching." Such sensations, arising from the normal skin, closely resemble a modified and controlled form of the widely diffused reaction, caused by brushing the hairs in this stage of recovery. These impulses travel up the spinal cord in conjunction with others of tactile origin. For, if all sensibility to pain, heat, and cold is destroyed in consequence of an intramedullary lesion, but touch remains unaffected, tingling and itching can still be produced by stimulating the hairs, and the sole can be tickled so as to evoke either pleasure or discomfort.

Throughout their whole course, within the limits of the spinal cord, tactile impulses are associated with the power of localizing the position of the spot touched. All recognition of the posture of the limb in space may be lost, and discrimination of two points may be impossible, but localization will not be affected, unless tactile sensibility is disturbed. But, above the nuclei of the posterior columns, tactile impulses tend to separate from those which underlie the power of appreciating the situation of the stimulated spot. The long connexion of topical localization with the integrity of one or other form of tactile sensibility is then broken for the first time.

§ 4.—*Other Spacial Aspects of Sensation.*

When the sensory supply of a part of the body consists of the deep afferent system only, the patient can recognize its position in space, and can estimate the direction and extent of passive movements as certainly as on the normal side. Complete division of all cutaneous nerves to a finger in no way affects the power of appreciating relationships in three-dimensional space.

The presence of deep sensibility only is sufficient also to endow the affected area with remarkably accurate localization, provided sufficient pressure is used to induce a sensation of contact. But tactile discrimination is impossible; the patient cannot distinguish two points of the compasses, when separated to the farthest distance within one segment of the limb.

Thus, the deep afferent system gives origin to all the impulses which are concerned with three-dimensional recognition and to many of those responsible for accurate localization of the stimulated spot; but it plays no part in the appreciation of tests in two-dimensional space. When we consider how closely this aspect of sensation depends upon impulses from the surface of the body, it is not difficult to understand the reason for this peculiar dissociation.

Tactile discrimination is closely bound up with the integrity of the epicritic system. During the protopathic stage the reaction to contact is massive and diffuse; it is impossible to indicate accurately the position of the stimulated spot, and two points cannot be distinguished. Contacts with two points of the compasses simultaneously, or with one point only, are confused and the sensory response bears no obvious relation to the single or double nature of the stimulus.

One of the earliest signs of returning epicritic sensibility is the appearance of tactile discrimination. At the same time, the reaction becomes less diffuse, and sensations are no longer referred to remote parts of the affected area. One of the most potent methods by which the presence of epicritic sensibility controls the vehemence of protopathic reactions depends on this recognition of two-dimensional relationships. The widespread reaction is not only inhibited by the impulses underlying tactile discrimination, but the ultimate sensation is restricted approximately to the area stimulated. As soon as this direct relation between the superficial extent of the stimulus and the sensory reaction is established, it becomes possible to compare the relative intensity of two external stimuli. A tube containing water at 20° C. is no longer thought to be "colder" than a metal rod of considerably smaller superficial area at the temperature of ice; for this error of appreciation depends on the more massive reaction evoked by the larger of the two stimuli, which renders impossible any accurate comparison of their relative intensity. Epicritic sensibility controls the diffuse "all or nothing" protopathic reaction in two ways. It endows the afferent mechanism with the power of adaptation, so that its response depends not only on the sensory quality of the stimulus, but also on the previous experiences to which it has been recently exposed. Secondly, it gives to the affected area the power to respond to relationships in two-dimensional space, and probably in this way curbs that radiation and reference into remote parts so characteristic of the protopathic response.

Tactile, painful and thermal impulses ultimately cross to the opposite side in their passage through the spinal cord and as soon as they have passed into secondary tracts show evidence of recombination. But those which underlie spacial recognition in two and three dimensions do not cross, until they reach the nuclei of the posterior columns at the upper end of the spinal cord.

Experimentally, this is a most fortunate condition, for it enables us to study the changes in sensation produced by the destruction of these spacial elements, in parts of the body otherwise of normal sensibility. An injury, confined to one half of the spinal cord, may cause loss of power to recognize posture and passive movement, together with want of discrimination of the compass-points on the side of the lesion, whilst every other disturbance of sensibility it may produce will be found on the opposite half of the body. We can therefore investigate the nature and consequences of this defective spacial recognition, unhampered by any other concomitant sensory loss; and, if the lesion lies sufficiently high in the cervical region to affect the hand, we can test its effect upon the appreciation of size, shape and weight.

Head and Holmes ([4] p. 110) have described such a case of lesion of the second cervical segment, the highest point that can possibly be affected within the limits of the spinal cord. The patient was at first paralysed in the right arm and leg; but this passed off, leaving him capable of every movement, though somewhat weaker in the right hand than in the left. When he came under observation two years later, the deep reflexes in the right arm and leg were exaggerated and the right plantar gave an upward response. Motion and reflexes were completely unaffected on the left half of the body, and it is obvious that the lesion must have occupied the right half of the spinal cord only.

As is usual in such cases pain, heat and cold were not appreciated over the whole of the opposite half of the body. All forms of tactile sensibility were however completely preserved, the position of any part of the left upper or lower extremity could be recognized with ease and passive movements were appreciated within normal limits. The compass-points could be accurately distinguished and topical localization was perfect.

When, however, we turn to the condition on the right half of the body, the side of the lesion, the condition was almost the exact opposite. Sensibility to touch, pain, heat and cold was perfectly preserved, but the patient was unable to recognize the posture of his right arm and leg, and could neither name nor imitate correctly the positions into which they had been placed. The compass-points could not be distinguished on the right palm, even at a distance of 6 cm. from one another. All power of recognizing size, shape and form in three dimensions was lost in the right hand, in spite of the fact that tactile sensibility was perfectly preserved. When some common object, such as a key, a coin, or a pencil, was placed in his right hand he was unable to say what it was. He could not estimate weight, nor recognize the relative hardness or softness of things held in the right hand.

In this case the power of appreciating relations in two- and in three-dimensional space was grossly disturbed on the right half of the body, whilst "spot-finding," or one-dimensional localization, remained unaffected. Within the limits of the spinal cord, this faculty is still linked up with impulses underlying the qualitative aspects of sensation which finally cross to the opposite half of the spinal cord. But those responsible for postural recognition and the discrimination of two contacts remain uncrossed in the posterior columns, throughout the whole of their course within the spinal cord.

Now it is obvious that these columns consist mainly of projections from the peripheral nervous system. No synaptic junction guards their entrance and none of their exogenous fibres cross to the opposite side, as is the case with every secondary sensory path. They are not specific tracts and, from the functional aspect, do not belong to the intramedullary system. Every afferent impulse, which enters the spinal cord, travels for a longer or shorter distance in the posterior columns. Some, like those capable of exciting sensations of pain, of heat or of cold, are rapidly combined and pass away into specific tracts on the opposite half of the spinal cord and brain-stem; they run, without further interruption, to end in the receptive centre of the optic thalamus. Others, like those concerned with sensations of contact, seem to follow a double path. Finally, however, of all the afferent impulses, which entered some distant segment of the spinal cord, none remain in the posterior columns, except those which underlie recognition of changes in two- and three-dimensional space. At the extreme upward termination of the spinal cord, these pass into the posterior column nuclei and cross to the opposite half of the nervous system. Up to this point, they have remained impulses of the primary level, which happen to have formed more or less qualitative groups in consequence of the gradual passing away of their peripheral companions; in the posterior column nuclei, however, they undergo their first re-grouping and travel on in specific sensory tracts ([8] p. 636).

Above the posterior column nuclei, impulses underlying the appreciation of posture and passive movement become separated from those concerned with tactile discrimination. These two aspects of spacial sensibility may be completely dissociated. Head and Holmes ([4] p. 120) described a case of a lesion in the brain-stem where recognition of passive movement was disturbed in the hand of the opposite side, whilst the compass-test gave identical results on the two halves of the body; the appreciation of size, shape and form in three dimensions was entirely unaffected.

All afferent impulses, which reach the optic thalamus, pass through the receptive portion of this organ on their way to their final sensory centres. But by the time they reach this junction in the nervous system, they have become arranged into physiologically specific groups: those associated with sensations of touch, of pain, of heat and of cold run in specific tracts to terminate in separate thalamic end-stations.

Such a mechanical arrangement is eminently comprehensible; for each of these categories corresponds to some aspect of sensation, which can be discovered by introspection. But our account of the arrangement and course of the impulses, which are concerned with spacial recognition, must seem entirely unreasonable to the *a priori* psychologist. As soon, however, as our conclusions are looked at physiologically, all difficulty disappears; for the method by which such impulses are integrated, on the physiological level, is admirably adapted to ensure the part which they must finally play in sensation.

An appreciation of the sensory aspect of movement in three-dimensional space depends on impulses arising in the deep afferent system, whilst the faculty of discriminating two or more points of contact on a flat surface is associated with the integrity of end-organs in the skin. The fibres of these two peripheral mechanisms are continued centralwards in the posterior columns of the spinal cord. These tracts are not, functionally, a portion of the central nervous system, as far as their exogenous fibres are concerned, and the impulses underlying spacial appreciation travel independently along each of the central projections of the two peripheral mechanisms in which they arose. They meet their first sensory synaptic junction in the cells of the posterior column nuclei. Here an opportunist anatomical arrangement becomes converted into a definite functional dissociation and, from this point onwards, each group of impulses travels in a separate specific tract.

Meanwhile, however, the long association of "spot-finding" with contact sensibility is also broken; one-dimensional localization becomes separated from the power of recognizing touch and pressure.

It is easy to see how this intimate connection, between the impulses underlying recognition of contact and of its point of impact, came about on the primary level. They arise simultaneously from the action of almost every external stimulus on the same two peripheral systems; the tactile aspect of pressure, and the power of appreciating the spot to which it is applied, are a function of the deep afferent mechanism, whilst recognition of the finer degrees of touch and their accurate localization depend on impulses from the epicritic system on the surface of the body. First of all, integration must weld the physical substrata of these two aspects of contact sensibility and localization into one coherent physiological group. This occurs in the secondary paths of the spinal cord and brain-stem. But, when we consider sensation from the psychical point of view, its spacial relations are indivisible; localization forms an inseparable part of our appreciation of space. The impulses upon which it depends must, therefore, become detached from those underlying sensations of contact, in order that they may be combined into one coherent spacial group. Finally, all those impulses potentially capable of revealing spacial relations impinge upon the appropriate centre in the cortex of the brain, to give birth to those processes which immediately underlie the sensory aspects of space. This is an example of the rule, that the arrangement of the peripheral nervous system depends on structural and developmental conditions; that of the intramedullary level is essentially functional or physiological, whilst the final processes, which underlie sensation, are grouped according to categories that can be discovered by introspection.

§ 5.—*Sensory and Non-sensory Afferent Impulses.*

When a tract degenerates in a centripetal direction, it probably conducts afferent impulses, but it is not, therefore, of necessity sensory in function. Some afferent impulses are destined never to affect consciousness, whilst others may or may not form the basis of a sensation, according to circumstances. This distinction has not been sufficiently emphasized, but is the key to many otherwise inexplicable arrangements in the central nervous system.

Thus, the posterior columns of the spinal cord conduct, amongst other impulses, those from the deep structures such as joints, tendons and muscles; on these depends our ability to recognize the position of the body in space. But, apart from this conscious recognition of posture, it is of supreme importance that the great centres for the maintenance of posture and bodily tone should receive afferent impressions from the joints. Impulses underlying deep sensibility, therefore, act upon the cerebellum and its allied centres, and in so doing remain for ever outside consciousness.

The afferent stream from deep structures, passing up the posterior column of the spinal cord, can affect two terminal centres. One of these is the cortex, with consequent recognition of posture and movement; the other, the cerebellar system, regulates and controls the postural and tonic aspects of muscular activity without in any way exciting consciousness directly. We know that some part of our body has assumed a certain position, or that a definite movement is taking place under the influence of the will; but the preliminary co-ordination of muscle-groups and shifting of tonic innervation, necessary for such changes, are carried on, without the accompaniment of any conscious process.

This is shown by the complete absence of sensory disturbance in uncomplicated lesions of one half of the cerebellum. Motion is gravely affected; the patient is unable to make rapid rotatory movements, or to touch his nose readily with the affected hand. He may titubate, or deviate grossly, when attempting to walk round a table and be unable to stand on the affected leg, even when his eyes remain open. And yet the closest examination fails to reveal any want of recognition of posture, or passive movement, loss of vibratory sensibility, or other analogous defect.

Holmes ([9] p. 512) has given a striking example of the difference in this respect, produced by lesions of the cerebellum and of the cerebral cortex, and has incidentally exposed a fallacy in the deductions of other observers. He showed that, if a patient with a unilateral lesion of the cerebellum was told to compare two identical weights placed simultaneously one in each hand, the weight on the affected side was thought to be the heavier. Even when they differed from one another by one quarter (200 to 150 grm.), the lighter weight might seem to be heavier on the same side as the lesion. But this defect is not primarily one of sensation, for, if the same two weights were placed one after another on the affected hand, they were uniformly estimated correctly. When the patient was compelled to compare one weight, supported by a limb whose movements were inco-ordinate, with an identical or even heavier weight on the normal hand, he tended to overestimate the one on the affected side. But, if he was allowed to use the same defective mechanism to weigh both objects, he was able to gauge their relation as accurately as on the normal palm.

I can fully confirm these observations from experiments made upon a patient under my care with injury to one half of the cerebellum. Equal weights placed simultaneously in the two palms were invariably said to be heavier on the affected side. It was not until the relation became that of 100 to 40 grm., that he thought the two weights were equal. But, if they were placed one after the other on the affected palm, he had no difficulty in giving uniformly correct answers, even when they differed so little as 100 grm. and 80 grm. This was also the limit of his appreciation on the normal hand, and many healthy persons cannot even reach this standard of accuracy. The following experiment shows how completely the original failure to estimate weights, placed simultaneously on the two hands, was due to a disturbance of movement in the affected limb. When the two hands were completely supported and weights were placed one in each palm, he had no difficulty in recognizing their identity, or the difference between 100 and 80 grm., provided he made no movement. That is to say, he could estimate correctly the relative pressure of the two weights, provided the muscles were at complete rest; but as soon as the hands were lifted, this perfect recognition was disturbed by the inco-ordinate movements in the limb on the side of the lesion, and he thought the weight on the affected hand was the heavier.

Throughout their passage within the posterior columns of the spinal cord, impulses concerned with the regulation of posture and movement are given off into direct cerebellar paths on the same side of the body. This is probably a reason why such afferent products of deep sensibility remain uncrossed, until they reach the nuclei of these columns at the termination of the spinal cord. By this time they have given off most of the impulses destined to influence unconscious postural activities, and the remainder become recombined and cross rapidly into secondary tracts of the brain-stem, to underlie conscious recognition of posture and movement.

Apart, however, from their unconscious effect in co-ordinating voluntary movement, these postural afferents exercise a profound influence on the character of spinal reflexes. When the cord has been gravely injured and the conditions are favourable to the return of activity in its severed portion, the response to stimulation sometimes assumes a massive and diffuse character. (Head and Riddoch [5]). The lower extremity becomes flexed, the abdominal muscles contract, bladder and rectum are thrown into activity and the patient may sweat over those parts of his body, which are innervated from below the lesion. Whatever the position of the limbs or the site of the stimulus, the answer is of the same character, widespread and uncontrolled. In order that this mode of reaction may appear, the mechanism for postural co-ordination must have undergone such gross injury, that no efferent impulses from the mid-brain centres can pass through the damaged region. Consciousness appears to play no essential part in determining whether it shall appear or not; for the patient may be insensitive to all forms of stimulation, however intense, and may be unable to appreciate the posture and movements of the lower extremities, and yet this massive response be absent.

It is to these unconscious postural and spacial impulses that we owe the adaptation of spinal reflexes to the site of the stimulus and the position of the limb. They act upon centres of the mid-brain and pons, and so control the reflex motor activity of the cord through proprio-spinal paths and centres. So long as this mechanism is intact, the great postural centres can dominate spinal reflexes. The answer to stimulation is endowed with local and postural signature. Not only the nature of the stimulus but its superficial extent and the position of the limb determine the character of the motor response.

In addition to these afferent impulses which never reach consciousness, there are a number of others which do not normally excite sensation. Thus, many impulses from the viscera produce an appropriate reflex action, without primarily affecting consciousness. When, in the course of some experiments on visceral sensibility, I passed warm water into the gut through a colostomy opening, the patient complained that he wanted to defæcate; a reflex peristalsis was set up, of which he was conscious, although he failed completely to recognize the stimulus by which it had been evoked. Most of the afferent impulses from the stomach and intestines probably belong to this order.

But many impulses capable of forming the basis of a sensation are prevented under normal conditions from reaching the highest centres;

or, if their forward path is not completely barred, they pass on in a profoundly modified form, in consequence of the concurrent activity of other sensory end-organs. The utility of this arrangement is obvious, especially in the case of those impulses which underlie discomfort or pain. Temperatures of from 40° to about 45° C. normally produce a pleasurable sensation of warmth; but, in the absence of the heat-spots and the epicritic thermal mechanism, such temperatures cause pain. This is well seen in the experiment on the glans penis, where a temperature which was exquisitely disagreeable became pleasant as soon as the warm water reached the area supplied with heat-spots.

It is obvious, therefore, that any temperature between 40° and 45° C. is an adequate stimulus to the pain-spots. Every time the hands are dipped into water between these degrees, innumerable impulses are evoked which are potentially painful. But they are prevented from affecting consciousness by the coincident excitation of the sensory heat-mechanism in the skin; for a sensation of pleasant warmth is incompatible with the co-existence of pain.

As the temperature rises, these potentially painful impulses increase in strength, until they can no longer be inhibited; they then form the basis of a sensation of pain. Here, consciousness is not disturbed until impulses are produced, not only in themselves of sufficient strength to evoke a sensation, but able to overcome the inhibitory effect due to the activity of other specific end-organs.

In a similar way, the return of epicritic sensibility reduces the amount of pain caused by cutaneous stimuli, without at the same time raising the threshold. Radiation and reference are inhibited and the pain produced by a prick is restricted to the immediate neighbourhood of the spot stimulated. This diminution in extent reduces the amount of pain suffered by the patient, although the measured threshold for painful sensations may be actually lower than during the preceding protopathic stage.

Pain is the oldest defensive reaction, and potentially painful stimuli are the basis of all primitive reflexes. It is therefore most important that potentially painful impulses should be diminished in extent, or actually inhibited in favour of those sensory impressions more capable of leading to discrimination. The mechanism underlying the production of pain must however remain in full physiological activity, so that it may play its part in defence of the organism, when noxious stimuli reach a high rade of intensity.

Thus, we have been able to show that the existence of non-sensory afferent impulses plays an important part in determining the mechanical co-ordination of voluntary action and are also of paramount importance in the production of spinal reflexes. Moreover, the evolution of the more discriminative functions of sensibility is largely bound up with the control of afferent impulses, which, if they affected consciousness, would evoke sensations of pain and feelings of discomfort.

§ 6.—*Feeling-Tone and the Optic Thalamus.*

Anatomically, the optic thalamus is one of the most intricate regions of the central nervous system; and, since most pathological conditions in this situation do not produce selective destruction, the clinical picture presented by a thalamic lesion is usually extremely complex. But, by a suitable choice of illustrative cases and by analysis of the symptoms they present, it is not difficult to discover the part played by this organ in sensation. For first of all, it is an end-station for all sensory afferent impulses, whence they start on two main lines to culminate in centres, which subserve two diametrically opposed aspects of sensation.

I shall not enter into the complexities of a case of thalamic disease; these are fully dealt with in the paper by Head and Holmes [4], where many illustrative examples are given. My business here is to show the fate of those afferent impulses, ultimately destined to excite the activity of the cerebral cortex and so endow sensation with certain specific qualities, dependent for their existence on this centre. But it is necessary first to consider how the optic thalamus influences the general stream of impulses, which travel onwards from its receptive end-station.

Above this junction in the nervous system, afferent impulses have the opportunity of affecting two sensory centres: on the one hand, the essential organ of the optic thalamus, distinct from its receptive mechanism, and on the other the cerebral cortex. This essential organ of the thalamus is the centre of "awareness" for certain aspects of sensation. It responds to all stimuli capable of producing sensations of a change in state. Thus, contact, especially movement across the surface of the body, arouses in the patient's mind recognition that something is happening, although he may be unable to appreciate the quality or situation of the stimulus. The thalamic centre reacts to the affective aspect of sensibility; when the patient exclaims, "Something is happening to me, I am being hurt," instead of, "You are sticking a pin into me," he is emphasizing the consequences of such thalamic appeal.

The essential organ of the optic thalamus is the centre for this aspect of sensation and responds to all stimuli capable of evoking either pleasure or discomfort. The feeling-tone of somatic or visceral sensations is the product of thalamic activity, and the greater the affective reaction to any stimulus the more certain is its thalamic appeal. Conversely, the more entirely any aspect of sensibility depends upon discrimination, as for instance the recognition of spacial relations, the less does it call upon the activity of the optic thalamus.

The sensory impulses evoked by a glass of hot water, held in my hand, influence my consciousness through these two centres. Recognition of contact, the sensation of heat and the pleasure or discomfort it gives me, according to the degree of temperature, are the product of thalamic activity, whilst the roundness, size and weight of the containing vessel, together with my power of appreciating the posture of the hand in which it lies, are the cortical aspect of the resultant general sensation.

During the passage through the secondary tracts of the central nervous system, sensory impulses undergo integration into groups of similar physiological significance. By the time they leave the receptive organ of the optic thalamus, this recombination on functional lines is complete, and, from this point onwards, sensory impulses travel in seven separable streams, each of which may be interrupted independently of the other.

The grouping at this level is still strictly physiological; each set of impulses arising from a certain aspect of peripheral stimulation still carries with it, not only the power of evoking a specific sensation, but also the capacity of endowing it with the appropriate feeling-tone. From the thalamic junction onwards, these physiological combinations have the opportunity of acting on two terminal centres. They can excite the essential organ of the optic thalamus, which is associated mainly with the affective side of sensation, or the so-called "sensory cortex," the centre for its more discriminative aspects. Each of these two end-stations reacts to the afferent quality to which it is attuned, and refuses to respond to those with which it is not in harmony.

How this selective reaction works in practice will be evident, if we consider in detail the constituents of each of these seven afferent streams.

(*a*) Tactile elements: This group contains all those impulses evoked by every form of contact, superficial and deep; they form a graduated scale from the lightest touch to the heaviest pressure, and include the physiological effects of moving rough objects across the surface, of stimulating the hairs, and of all manipulations which cause tingling and itching.

Now it is obvious that these impulses must make a double appeal. Sudden contact with some hard body, a rough object dragged across the skin, brushing the hairs and tickling stimuli are all sufficient to excite the thalamic centre; the sensations they evoke are heavily charged with feeling-tone. But a fine appreciation of the different degrees of tactile stimuli, or of the pressure of various weights placed on the fully supported hand, makes an appeal to the discriminative aspect of sensibility with which the optic thalamus has no concern.

(*b*) Painful elements: Here we find a condition the exact converse to that just described for the tactile elements. All painful or uncomfortable stimuli appeal mainly to the thalamic centre and, slightly if at all, to that in the cortex. But it is necessary at the outset to distinguish clearly between "discomfort" and "pain." Pain is a distinct sensory quality equivalent to heat and cold, and its intensity can be roughly graded according to the force expended in stimulation. Discomfort, on the other hand, is that feeling-tone which is directly opposed to pleasure. It may accompany sensations not in themselves essentially painful, as for instance that produced by tickling the sole of the foot. The reaction produced by repeated pricking contains both these elements; for it evokes that sensory quality known as pain, accompanied by a disagreeable feeling-tone, which we have called discomfort. On the other hand, excessive pressure, except when applied directly over some nerve trunk, tends to excite more discomfort than pain, and makes a more exclusively thalamic appeal.

The secondary tracts in the spinal cord, devoted to the painful aspects of sensibility, conduct all impulses, capable of arousing pain or discomfort, in connection with injuries to the superficial or deep parts of the body. But even before the fillet actually terminates in the optic thalamus, the more disagreeable stimuli, such as excessive pressure, can influence consciousness, though the affected parts are totally insensitive to the pain of pricking or burning. This is evidently the first indication of the ultimate action of all painful and uncomfortable stimuli on the affective centre in the optic thalamus. But, although the appeal of all such aspects of sensibility is overwhelmingly thalamic, it must not be forgotten that pain can be roughly graded according to the intensity of the stimulus; in so far, it is the product of cortical activity. How small is this element in most disagreeable stimuli is shown by the extreme difficulty in devising graduated painful tests, which are capable of giving measurable results.

(*c*) and (*d*) Thermal elements (heat and cold): The impulses underlying sensations of heat and cold travel up to the spinal cord and brain-stem in specific tracts, and still remain separable above the level of the thalamic junction. Each of these afferent groups can exert a profound influence, both on the essential organ of the optic thalamus and on the sensory cortex. Cold is almost universally uncomfortable, but heat may excite either pleasure or discomfort, according to the temperature of the stimulus; feeling-tone is always an important characteristic of thermal sensations.

But, apart from the affective aspect, we believe that the optic thalamus forms the physiological end-station for those activities which underlie the qualitative appreciation of thermal stimuli. A patient, in whom the sensory cortex has been thrown out of action, may recognize heat and cold correctly, although he is unable to appreciate the relation between two hot or cold objects. Crude sensations of heat and of cold are functions of the activity of the essential organ of the optic thalamus.

On the other hand, finer discrimination of thermal stimuli is a function of the cortex. The normal human being can distinguish different degrees of temperature with considerable accuracy, although they do not differ in quality from one another. Both 35° C. and 42° C. are said to be "warm," when applied to a sensitive part, such as the palm of the hand, but there can be no doubt that the latter is the warmer. This power of discriminating between two stimuli of the same sensory quality becomes less accurate the nearer they approach to the extremes of heat or of cold; for, at the two ends of the scale, the reaction tends to approximate to the "all or nothing" response, so characteristic of the activity of the heat- and cold-spots.

Thus, the optic thalamus forms the physiological end-station for those aspects of thermal sensibility concerned with feeling-tone and crude qualitative appreciation of heat and cold. The sensory cortex, on the other hand, is responsible for the faculty of discriminating quantitative differences in intensity.

(*e*) (*f*) and (*g*) Spacial elements.—Above the level of the posterior column nuclei, impulses concerned with the three aspects of spacial recognition run in three separate streams, apart from those associated with sensations of touch and pressure. There is no evidence to show that these groups make any thalamic appeal. They are associated with purely discriminative processes, such as the power of appreciating posture, recognition of two or more simultaneous contacts and localization of the stimulated spot.

On the integrity of each of these afferent groups depends the faculty of responding accurately to some more complex test. Thus, the group concerned with relations in three-dimensional space is not only responsible for our recognition of posture and passive movement, but also, at this level, endows us with the power of estimating correctly the difference between two weights placed on the unsupported hand one after the other. The act of "weighing" an object in the palm is a function of this stream of impulses.

In the same way, appreciation of the relative size and shape of flat objects depends on the same afferent group as the power of discriminating the two points of the compasses applied simultaneously.

Thus, in conclusion, we have been able to show that afferent impulses pass on from the receptive centre in the optic thalamus in seven separable streams. Each of these acts upon one or both of the terminal physiological centres. At the one pole, stand the effects produced by painful stimuli, which profoundly affect the essential organ of the optic thalamus, but have little influence on cortical activity; at the other extreme we find the physiological substrata of spacial recognition, which appeal exclusively to the sensory cortex.

§ 7.—*Discrimination and the Cortex Cerebri.*

The paths between the termination of the fillet in the optic thalamus and the essential thalamic centre lie in the body of this organ and cannot be completely interrupted without producing some destruction of incoming sensory impulses or injury to the centre itself. But the paths from the receptive end-station in the optic thalamus to the cortex pass by way of the internal capsule and the radiating fibres of the centrum ovale. These tracts are frequently interrupted by disease, and it is therefore possible to determine the nature and grouping of the sensory impulses, which they conduct to the cortical centres.

Lesions of these subcortical paths show that we are still dealing with physiological processes destined to excite those central activities which underlie the cortical aspects of sensation; the defects of sensibility are evidently due to the fact that certain afferent elements fail to reach the intact centres of the cortex.

We have described, in the previous section, the groups of afferent impulses which pass away from the terminal receptive mechanism in the optic thalamus to influence, on the one hand, its essential centre, and on the other the sensory cortex. Subcortical lesions reveal the same streams, differing only in the fact that they have shed all those elements capable of exciting thalamic activity. Thus, as before, each of the three groups of spacial elements seems to run independently of the others, and can be affected separately. Moreover, a loss of power to recognize posture and movement still carries with it inability to compare the weight of the two objects, placed successively on the unsupported hand, and defective discrimination of the compass-points is associated with want of appreciation of relative size and shape in two dimensions.

At this level of the nervous system, an interference with tactile impulses does not prevent the patient from recognizing contact or roughness. But it disturbs appreciation of the relative intensity of graduated tactile stimuli and of increase or diminution in weights placed on the fully supported hand.

Any disturbance that may be present in thermal sensibility is shown by defective recognition of the relation between temperatures of the same quality. The patient can still distinguish heat and cold, but cannot be certain if one warm object is warmer or cooler than another. At the same time, the neutral portion of the scale, where the stimulus seems neither hot nor cold, may be considerably enlarged. That is to say, any actual loss of sensation is found amongst those temperatures, which must be compared to be appreciated and are most susceptible to sensory adaptation.

Such are the materials out of which the cortex manufactures those physiological processes, which underlie certain specific aspects of sensation. When, however, we examine the sensory effects produced by a lesion of the cortex, we are no longer confronted with a gross physiological defect, but with what appears to be a want of attention; the patient seems to be untrustworthy. Sometimes he appreciates a touch, sometimes even a stronger stimulus fails to evoke an answer; he knows that the object in contact with the affected part is warm, but cannot tell whether it is hotter or colder than one he has previously held in his hand. Sensation is not abolished, but his answers are confused; and yet on further examination they are found to be unsatisfactory only in so far as they concern the affected part. Elsewhere, he may give results which in accuracy are up to or even above the normal average.

Analysis of this apparent untrustworthiness of the affected part shows that it depends upon three main factors. First of all, the answers given by the patient are inconstant; at one moment he seems to appreciate a stimulus of a certain intensity, at the next it remains entirely without effect upon his consciousness. Thus, it may be necessary greatly to increase the stimulus before a series of correct answers can be obtained, and not infrequently even then no regular response can be evoked. In such a case, we cannot work out a threshold, not because the patient never appreciates the stimulus, but because at every intensity it sometimes causes a sensation and sometimes passes unnoticed.

Secondly, a cortical lesion tends to destroy the power of appreciating the difference between two objects which have certain basic sensory qualities in common. Thus, the patient may no longer be able to tell which of two weights is the heavier, although they are of the same shape and cover an identical surface. He may also lose the power of recognizing the relative size and form of objects and, although he can tell whether a fabric is rough or smooth, he may no longer be able to distinguish flannel from silk.

Thirdly, the cortex is responsible for the spacial aspects of sensation and one of the most frequent consequences of a lesion in this situation is inability to recognize correctly the posture of the affected parts of the body; this is often accompanied by a more or less defective discrimination of the compass-points and some want of power to determine the situation of the spot touched. But these changes in sensibility are curiously uncertain. A varying reaction to constant stimuli, hallucinations, and wrong answers of all kinds mar the measured records to an extent not usually present when the lesion lies at lower levels of the nervous system.

The cortex is the repository of past impressions and these sensory dispositions profoundly modify the effect produced on the arrival of fresh impulses. As far as the spacial aspect of sensibility is concerned, a disturbance of these sensory dispositions would be responsible for just that uncertainty of recognition of three-, two- and one-dimensional relationships, which actually follows a cortical lesion. For it would be impossible to discover the position of any part of the body, unless the immediate postural impressions were related to something that had preceded them. A direct perception of posture, analogous to that of roughness, cannot occur; in every case, the new position of the limb is related to some previous posture.

We have been able to show that the standard against which a change in posture is estimated is not an image either visual or motor; it lies outside consciousness. Every recognizable change in posture enters consciousness already charged with its relation to something which has gone before, and the final product is directly perceived as a measured postural change. For this combined standard, against which all subsequent changes in posture are estimated, before they enter consciousness, we have proposed the word "schema." Man perpetually builds up a model of himself, which constantly changes. Every new posture or movement is recorded on this plastic schema and the activity of the cortex brings every fresh group of sensations evoked by altered posture into relation with it. Immediate postural recognition follows as soon as this relation is complete. A cortical lesion tends to destroy such schemata and so disturbs the certainty of spacial recognition.

Inconstant recognition, want of appreciation of intensity and inability to relate and measure sensation are the consequences of a lesion in certain portions of the cortex. Attention can no longer move with freedom and certainty over the objects presented to it, so far as they arise in consciousness connected with sensory processes from the affected parts of the body. Obviously then, the consequences of a pure cortical lesion produce changes of which the manifestations are essentially psychical. We are above the level at which the defects in sensibility are due to interference with afferent impulses and the disturbances produced by a cortical lesion are manifested in defective attention and discrimination.

CHAPTER III.—THE METHOD AND AN ILLUSTRATIVE CASE.

§ 1.—*The Method.*

Such were the views we expressed on the activity of the cortex in the production of sensation. We had determined the manner in which the affected parts of the body reacted to a series of sensory tests, and were anxious to demonstrate the general afferent functions of the cortex, unhampered by an analysis of dissociated sensibility or questions of anatomical localization.

But it is obvious that our tests were empirical, and, though each was selected to explore some special aspect of sensibility, we were not justified in correlating the numerical results yielded by one test with those of another. Even the three methods we used for estimating defects in spacial recognition could not be directly compared. Suppose, for example, that passive movements of less than 30° in extent were not appreciated, and the compass-points could not be discriminated until they were separated by a distance of 3 cm.; how can we say which of these two aspects of spacial sensibility was disturbed to a greater degree? Both were obviously defective, but we cannot compare the relative amount of the affection.

Still less can we correlate directly the numerical results of tests depending on sensory impulses of a different order, such as those underlying spacial recognition or the appreciation of thermal differences. Yet before it is possible to analyse the part played by the cortical centres in sensation, it is essential to find some method of correlating the amount of sensory loss revealed by each test in turn.

Now Holmes and I had already seen cases, such as those described by Russel and Horsley [14], where the condition of the hand was not uniform. Some digits showed gross loss of sensation, others were normal or but slightly affected. Not only might the severity of the loss differ over each finger, but its character might not be the same even on neighbouring digits. When, for instance, the little finger showed gross loss of recognition of passive movement, of tactile discrimination and of localization, it by no means followed that all these functions were disturbed on the ring-finger. It was obvious that such cases gave us the opportunity of determining the forms which sensory dissociation might assume when caused by a lesion of the cortex, and thus, incidentally, of grouping the main factors in its sensory activity.

For the more acute and severe the lesion, the wider will be the disturbance of function it produces, as far, at any rate, as the sensory cortex is concerned. This law holds, not only for sensation in its entirety, but also for any one specific aspect of sensibility; thus, in any particular case, one form may be disturbed over both hand and foot, whilst some other sensory quality is affected on the hand alone. Here we should be justified in saying that the more extensive loss signified a graver affection of the corresponding aspect of sensation.

The truth of this law will be abundantly proved by the many examples of sensory dissociation, which I shall consider more in detail later. But it is obvious that, in the light of such a hypothesis, cases where the various digits show different sensory dissociations become of fundamental scientific interest. Suppose, for instance, that one of the spacial aspects of sensibility were affected in four out of the five digits, whilst the power of appreciating graduated tactile stimuli was disturbed in the little and ring-fingers only, we are justified in assuming that the lesion has produced a greater loss of spacial than of tactile appreciation. By determining the number of digits, which are shown to be affected to each sensory test, we are able to correlate the disturbance of the various aspects of sensation.

Once this relation has been established, the actual results of measurement can be roughly calibrated and compared. We may find that on one of the digits every form of spacial recognition is grossly affected, but on the neighbouring finger the three-, two-, and one-dimensional tests give different results. Passive movement may not be recognized with certainty, however extensive the range within normal limits, whilst the two compass-points can be accurately discriminated if they are 3 cm. apart; "spot-finding," however, is not measurably disturbed, but the patient complains that it is more difficult to discover the exact point of contact than over similar parts of the normal hand. Here, then, on the same finger are three grades of loss. It is obvious that the three-dimensional test is grossly affected, because no threshold can be obtained. Discrimination of two points shows a smaller disturbance, for a threshold could be determined, although it was raised considerably above the normal. On the contrary, one-dimensional recognition was not measurably disturbed; but the patient knew by introspection that it was more difficult than over unaffected parts.

In civilian practice these cases occur so infrequently that it was impossible to investigate the problem systematically on these lines. But, as a consequence of the War, every neurologist has come across patients with injuries of the skull who have complained that one part of the hand "feels different" from the other. This may be a temporary phase on the road to recovery, or may remain almost unaltered for many months or years. I believe that these cases give the key to the part played by the cortex in the production of sensation, and enable us to guess at the topographical localization of the various sensory activities which they reveal.

Twenty-one such cases have come under my observation, and I have fortunately been able to work them out fully, in spite of the considerable expenditure of time necessary in each instance to obtain a complete set of records.

The methods I have used are those set forth in the paper with Gordon Holmes [4]. I have employed them systematically, because I knew their capacity and the fallacies to which they were exposed; moreover, it seemed better to investigate each of these remarkable cases on lines that would allow of correlation with civilian experience.

§ 2.—*An Illustrative Case.*

It will be well to begin with an account of one of these cases, so that the clinical phenomena they exhibit may be clear before we pass on to a more detailed examination of the sensory dissociation in each particular instance.

Case 17.—Private F. L., aged 29. On February 17, 1915, he was hit by a bullet which glanced from a sandbag and struck the left side of his head. He walked to the dressing station and was sent on to a field ambulance, arriving within three hours from the time of the wound. He suffered a good deal from pain in the head but did not vomit or feel sick. The right arm was weak, and, although he knew what he wanted to say, he had difficulty in finding words to express his meaning.

On February 19, he was operated upon by Captain Browne, who made the following notes: "Gunshot wound of head. Exit and entrance wounds over left motor area. Wounds joined up and extended. Gutter fracture of outer table; much fissuring of inner table; blood clot; dura incised, clot under dura with extensive pulping of cerebrum. Right arm, motor paralysis. Right facial paralysis and affection of speech. After operation, face and speech improved."

On March 3, he was transferred to London; his condition was carefully worked out by Dr. Fearnsides, who kindly arranged that I should see him. After some time spent at a convalescent home, he came under my care at the London Hospital in July, 1915. The following account is based upon records taken by myself during the first fortnight of that month.

At that time, four months after the wound, I found him an extremely intelligent, quiet Yorkshireman, who had left school at 12 years of age in the sixth standard, and held a second class Army certificate.

The wound had healed firmly and now consisted of a pulsating depressed scar covering a defect in the bone of an irregular diamond shape. This was situated 15·5 cm. behind the nasion and 1·5 cm. to the left of the middle line. The area of bony loss measured 8·5 cm. antero-posteriorly and 4 cm. across at the widest part. The nasion-inion measurement was 34·5 cm. and the inter-aural line cut it 14·5 cm. behind the root of the nose (fig. 17).

Speech was entirely unaffected. He had not suffered from any form of convulsion or seizure. All headaches had disappeared several months before.

Vision was $\frac{6}{6}$ with either eye and the visual fields were normal. The discs showed no signs of past or present change. Hearing, smell and taste were unaffected.

The pupils reacted normally. Ptosis, nystagmus and ocular paralysis were absent. The face, tongue and palate moved well and equally.

The right wrist-jerk was slightly greater than the left, but both were brisk. Knee-jerks, ankle-jerks and plantar reflexes were absolutely normal. Both upper and the left lower abdominal reflexes were brisk; but that from the right iliac fossa was defective. Scratching the subumbilical area on the right side produced a contraction above the level of the umbilicus only.

The only defects in motion were to be found in the right hand; with this exception, even the movements of the right upper extremity were performed powerfully against resistance and showed no inco-ordination.

The grasp of the right hand was fairly strong, but clumsy and considerably weaker than that of the left; it did not relax materially when his eyes were closed. If his hands were held out in front of him. the fingers were out of alignment and, when made to close his eyes, they fell into irregular movements of flexion and extension. The contrast between the falling away of the fingers and the steadiness of the arm and forearm was exceedingly striking. Even with the eyes open he could not flex or extend the thumb without coincident irregular movements occurring in the fingers, and no isolated movements were possible in any of the digits of the right hand. Told to flex the index finger, he bent it at once, but at the same time considerable flexion occurred in all the other fingers and at the terminal joint of the thumb. When asked to straighten the right index, the thumb became extended together with the terminal joints of the middle, ring and little fingers, although the metacarpophalangeal joints continued somewhat flexed. If, however, he was told to open all his fingers, he did so rapidly, though the fingers remained out of alignment, owing to a diverse amount of flexion remaining at the metacarpophalangeal joints. The amount to which the fingers could be extended varied greatly from time to time, but the little finger was always in the worst position.

These difficulties in movement were distinctly exaggerated when his eyes were closed. No gross increase or decrease of tone could be discovered in the fingers or thumb of the right compared with those of the left hand.

Sensation.

His only complaint on the sensory side was that his right hand "feels numb"; when he woke at night he knew he had a right hand, but it seemed stiff and dead.

Measured movement.—No sensory changes could be discovered to any of the tests we use, either in the upper extremity above the wrist, or in the lower limb. All deviations from the normal, found in this case, lay in the right hand and were worked out finger by finger with the following result:—

Measured movement	R. (affected)	L. (normal)
Thumb ..	1°	1°
Index finger	5° to 10°	1° to 2°
Middle finger	10° to 20° (gross loss)	1°
Ring finger	10° to 20° (gross loss)	1°
Little finger	10° to 20° (gross loss)	1° to 2°

In every case, at least ten observations were made on each digit, and it is evident that on the normal hand the patient gave a perfect series of answers with a movement through an angle of less than 2°. But on the right hand, the records from the middle, ring and little fingers showed gross defects of appreciation; not only was it necessary for the movement to range through from 10° to 20°, but the answers were uncertain and the direction in which the finger had been moved was not infrequently misinterpreted. In the case of the index finger, there was no such error or uncertainty, but the threshold was raised; a movement through from 5° to 10° was necessary for appreciation, whilst on the normal side flexion or extension of from 1° to 2° was at once recognized. No difference could be discovered between the range of movement necessary for correct appreciation in the two thumbs.

Vibration of the tuning-fork.—A tuning-fork beating 128 vibrations per second (C°) was placed on the palmar aspect of the terminal phalanx of the right little finger and allowed to remain there until the patient said it had ceased. It was then transferred to the similar part of the left hand and the vibration was appreciated for from ten to fifteen seconds longer. But, when the converse experiment was tried, he failed entirely to recognize any vibration in the affected little finger, after it had apparently ceased on the normal side. This result was recorded as follows:—

From R. little finger to L. + 15 sec. + 10 sec.
From L. little finger to R. + 0 sec. + 0 sec.

In the ring and middle fingers the records showed a less gross loss of sensation and in the index finger the loss was so slight as to be just perceptible only on the records.

From R. index finger to L. + 12 sec. + 11 sec.
From L. index finger to R. + 9 sec. + 8 sec.

There was no difference between the answers obtained from the two thumbs; evidently the right thumb was not affected to this test.

Simultaneous compass-test—

	R.	L.
Thumb	Perfect at 1 cm.	Perfect at 1 cm.
Index	Perfect at 1 cm.	Perfect at 1 cm.
Middle	Gross loss at 3 cm.	Perfect at 1 cm.
Ring	Gross loss at 3 cm.	Perfect at 1 cm.
Little	Gross loss at 3 cm.	Perfect at 1 cm.

To this test the little, ring and middle fingers were affected, whilst the index and thumb gave perfect records when the points of the compasses were 1 cm. apart. This is the smallest distance that can be safely used in clinical work; if the patient is unusually intelligent, it may be possible to obtain a perfect record at 0·5 cm., but such accuracy cannot be expected in the general run of cases.

With regard to the records from the little, ring and middle fingers, I have little doubt that the distance between the two points might have been considerably increased above 3 cm. without obtaining a threshold; for the patient did not show the slightest power of discrimination. But, when they are separated for much over 3 cm., one of the compass-points is liable to fall over the basal phalanx, which, in some of these cases, may be in a more normal condition than the two terminal ones.

Localization or "spot-finding."—There was distinct loss of the power to localize the spot stimulated, as shown by our modified Henri method, which avoids the fallacy of groping. Out of six tests applied to the palmar aspect of the right little finger, he made one erroneous localization, referring a touch over the middle phalanx to the middle of the basal phalanx. Moreover, he showed some hesitation even with those stimuli which were correctly localized; this was not the case on the little finger of the normal hand. Elsewhere, even over the affected hand, he was astonishingly quick and accurate. Thus we must conclude that the power of localizing correctly a single stimulus was somewhat disturbed on the right little finger, but was perfect elsewhere.

Measured tactile stimuli (the tactile hairs).—To this test the palmar aspect of the thumb, index and middle fingers of both right and left hands gave perfect readings with a hair of 21 grm./mm.^{2} But over the ring finger of the affected hand this hair was not uniformly appreciated and 23 grm./mm.^{2} was required before a perfect record could be obtained. That is to say, the threshold was here slightly raised.

On the right little finger the disturbance of sensibility was of a much grosser character, as shown by the following record:—

	R. (affected)	L. (normal)
21 grm./mm.^{2}		iiiiiiiiiiiiiii
21 grm./mm.^{2}	oiioiiioii ⁞ –oooioo	
23 grm./mm.^{2}	ooioioii–oii ⁞ ⁞⁞ –iii–ii	
35 grm./mm.^{2}	oiiiiii–iii ⁞ –iiiii–i	
70 grm./mm.^{2}	iiii–iii–iiiiii–iii	
21 grm./mm.^{2}	iii ⁝ –i ⁞ –oiii–oooi–oooo	
21 grm./mm.^{2}		iiiiiiiiiiiiiii

(An i signifies that he responded to the contact of a tactile hair of a strength given in the left hand column. An o records an absence of reply, whilst a dotted line represents an hallucinatory answer. The horizontal lines correspond to pauses of 5 sec. or more, interpolated either after a series of hallucinations, or in order to test the possibility that the previous answers might have been hallucinatory.)

Here the threshold was raised to 70 grm./mm.^{2} With all lighter stimuli, hallucinations and gross defects of response made their appearance.

Temperature.—It is always extremely difficult in cases of cortical lesions to obtain a trustworthy series of temperature records; for a large number of observations are necessary, comparing the behaviour of the two hands to the same temperature. This is impossible with a stupid patient and fatiguing to one who is intelligent, if it forms part of a long examination. In this case, therefore, I was satisfied with a careful exploration of the condition of the little finger of the right hand compared with that of the left.

For both hands the point at which he ceased to recognize the silver tube as cold was 27° C., whilst the threshold for heat was 30° C., the external temperature being 19° C. throughout these observations.

He made no mistakes in the relative warmth of silver tubes containing water at 35° C. and 45° C. throughout a series of tests on both little fingers.

In fact, were it not that he said, "It is never so clear on the right little finger as on the left," no abnormality could have been detected. Here there was evidently a diminution of sensory acuity so slight that it could not be measured, though it was evident to the patient himself. This I shall call an "introspective" difference.

Prick.—There was no difference between the two hands in the sensitiveness to a measured prick, tested finger by finger, and introspectively the patient was not aware of any abnormal sensations.

Weight.—The best method for testing the power of recognizing differences in weight in the five digits is to add and subtract leaden discs of the same surface area, but of different thickness. The hand is laid palm upwards on a pillow, in such a way that it is fully supported; a cork disc of negligible weight is laid on the palmar aspect of the finger under examination. To this, progressively heavier weights are added, and then removed until the cork alone remains on the finger. These weights are not added or removed in unbroken sequence, first up the scale and then down, but irregularly, and the patient is asked to indicate when any change occurs. He is required to say "on" or "off" only, or, if he prefers to do so, may answer "heavier" or "lighter." He is not allowed to raise the finger from its basis on the pillow so as to "weigh" the test object.

This test applied to the two little fingers gave the following records:—

Weight added or subtracted	R. (affected)	L. (normal)
+ 20 grm.	No reply	On
+ 40 ,,	On	On
− 40 ,,	On	Off
+ 40 ,,	On	On
+ 80 ,,	On	On
− 80 ,,	No reply	Off
+ 80 ,,	Off	On
+ 100 ,,	Off	Off
− 100 ,,	No reply	Off
− 80 ,,	No reply	Off
− 40 ,,	No reply	Off
− 20 ,,	No reply	Off
	Asked if any weight was on the hand he replied that it was still there	He then added "All gone."

A similar series of records was obtained from the right ring finger, whilst from the right index he gave a perfect series of answers.

Texture.—The test object, e.g., a piece of velvet, was passed round the top of each digit and then moved from side to side across the palmar aspect of the terminal phalanx. With the little finger of the left hand he recognized velvet, silk, cotton and flannel without difficulty, but failed entirely with the right; to each he said, "I don't know," or "I've no idea."

With the thumb and with the index finger of each hand he gave equally perfect answers.

The sensory condition of the various digits of the right hand revealed by these tests can be summarized in the following table :—

	Little	Ring	Middle	Index	Thumb
Measured movement	Gross loss	Gross loss	Gross loss	5° to 10°	Normal
Vibration	Gross loss	Affected	Affected	Slight loss	Normal
Compass-test	Gross loss	Gross loss	Gross loss	Normal	Normal
Localization	Uncertain	Normal	Normal	Normal	Normal
Textile hairs	Gross loss	Raised threshold	Normal	Normal	Normal
Temperature	Introspective	Normal	—	Normal	Normal
Prick	Normal	Normal	—	Normal	Normal
Weight	Gross loss	Gross loss	—	Normal	—
Texture	Loss	—	—	No loss	No loss

CHAPTER IV.—THE FORMS ASSUMED BY DISTURBANCES OF SENSATION DUE TO LESIONS OF THE CORTEX.

§ 1.—*Introductory.*

Before we pass on to consider the significance of this regional dissociation of sensibility, it will be well to summarize the conclusions which can be drawn from the case I have just described.

(1) Sensation as a whole may be affected by a cortical lesion to a different degree in the various digits. In this instance, the thumb was normal; but there was a steady deterioration in the sensory condition on passing towards the post-axial border of the hand, culminating in gross loss over the little finger.

(2) A similar tendency to graduated loss of sensibility was shown in the records of each individual test. Thus, the patient could appreciate passive movement equally well on both thumbs; but a range of 5° to 10° was required in the index finger, and gross loss was evident in the three other digits.

(3) Tests which appeal to fundamentally different aspects of sensation may not be affected over the same number of digits. In the case already described, appreciation of passive movement was defective in the index, middle, ring and little fingers, whilst the tactile hairs, a most delicate test, revealed loss of sensibility in the ring and little fingers only. This dissociation, as I shall show later, can go so far that one form of sensibility may be grossly defective whilst another may show little or no change.

(4) Even those tests which belong to the same sensory order may not be affected over the same number of digits. Thus, measured movement, the compass-test and "spot-finding," are all methods of exploring the spacial aspects of sensation. But, in the example I have chosen, recognition of movement was disturbed in four digits, tactile discrimination in three, and topical localization in one only.

Before we can draw any general conclusions from the detailed examination of these cases of dissociation, it is necessary to consider the nature of the sensory tests on which our results are based. None of them were invented as a special means of exploring the defects produced by a lesion of the cortex, and the majority excite to activity both the essential organ of the optic thalamus and the cortical centres. If we call to mind the various aspects of sensation which depend on the physiological activities of these two cerebral end-stations, we recognize that, whereas the optic thalamus deals with crude awareness to contact, heat, cold and pain, the sensory cortex endows these basic functions with spacial qualities, intensity and relativity.

When we pass in review, according to this conception, the sensory tests we habitually use, it is evident that recognition of passive movement, discrimination of two compass-points and our method of testing localization appeal to those elements in cortical activity underlying recognition of relations in space. Thus, measurement of the angle through which any part of the limb must be moved, in order that such change in posture may be appreciated, is an exploration in three-dimensional space. The power of discriminating two points of the compasses, applied exactly at the same moment, reveals the capacity for recognizing two-dimensional space, whilst "spot-finding" is an exercise in one-dimensional localization.

Further analysis on these lines shows that our methods for examining sensibility to touch, temperature and pain can be combined into another group from the point of view of the sensory cortex. For, although the basic response to all these tests is due to the activity of the optic thalamus, the power of recognizing differences of intensity in the sensation evoked is a function of the cortex. All the methods we use presuppose that an increase in strength of the physical stimulus will result in a sensation of greater intensity, in some more or less graduated proportion; on this principle depends the determination of a threshold, which plays so important a part in all our sensory examinations. But one of the signs of cortical disturbance is inability to obtain a threshold. The answers are uncertain, even to stimuli of the same strength applied in an exactly similar manner, and, although a response may be evoked, it is not of necessity in proportion to the degree of stimulation.

Of the remaining tests, weight is the only one applicable to the fingers which can be strictly graduated. As I have applied this test, it makes an appeal to another sensory faculty, the power of appreciating similarity and difference. At first it might appear to be only another means of gauging the recognition of intensity. But, if the methods of using the tactile hairs and graduated weights are compared, we see that the measure of tactility is given by the bending strain of the hair to which the patient responds; he is touched with one hair and gives uncertain answers, whilst a stronger hair may evoke constant recognition of a series of contacts. But, in testing with weights, one after another is placed on the hand, and he is then asked to say when a difference occurs; sometimes the load is made heavier by addition, sometimes lighter by subtraction. When this test is carelessly carried out, the act of removing or replacing a weight may arouse the patient's awareness and he at once responds; but he cannot tell with certainty whether the weight on his hand is lighter or heavier than it was before he received the sensation of "something happening." We are not dealing with his capacity to appreciate a contact of a certain intensity, but with his power of recognizing difference.

Yet, even after we have grouped our tests according to the particular aspect of cortical sensibility, which they reveal, it is necessary to remember that they differ greatly in the severity of the task they set to the patient, and in the accuracy of the answers they demand. It is far more difficult to recognize a minute range of passive movement in one of the fingers than to indicate, within necessarily wide limits, the situation of the spot stimulated. Similarly, a series of perfect answers to the contact of graduated hairs requires a more perfect reaction to differences in intensity than the crude comparison which is alone possible with a series of pricks.

For the sake of simplicity let us then select from each group of tests the one which gives the widest possible range of measured difference. The power of recognizing passive movement may be taken as the representative of the spacial aspect of sensibility; in the same way, the tactile hairs form the best measure for the faculty of appreciating graduated differences in intensity, whilst the addition and subtraction of weights can be taken as an indication of the ability to recognize similarity and difference in stimuli of the same sensory quality.

Case	Measured movement	Tactile hairs	Weights
No. 18	0	Affected (3 digits) ..	Affected (2 digits).
No. 3	Affected (5 digits) ..	0	,, (2 ,,).
No. 21	Whole hand affected ..	0	Defective.
No. 16	,, ,, ,, ..	0	Gross loss over whole hand.
No. 2	Affected (4 digits) ..	0 (or introspectively) ..	Introspective (1 digit).
No. 19	,, (5 ,,) ..	Slightly affected (3 digits)	0
No. 13	,, (4 ,,) ..	Affected (5 digits) ..	0
No. 22	,, (5 ,,) ..	Introspective (3 digits) ..	0
No. 14	,, (2 ,,) ..	Threshold raised (2 digits)	0

The cases collected in the above table show that it is possible for one or more of these representative tests to remain entirely unaffected, although a profound disturbance of sensation may be revealed by the remainder. Thus, in No. 18, recognition of passive movement was not affected in any of the digits in spite of a grave disturbance of tactility and of the power of responding to differences in weight. No. 3 showed no loss of tactile sensibility, and yet there was extensive want of appreciation of passive movement and of relative weights. No. 22 showed a profound loss to the tests for measured movement and an introspective defect to tactile stimuli, but perfectly appreciated the addition and subtraction of weights on all the fingers.

It is, therefore, probable that these three aspects of sensibility are due to distinct physiological activities, which are not uniformly distributed throughout the so-called sensory cortex; some are more intensely represented at one part, some at another. Otherwise it would be difficult to understand how one aspect of sensation could be gravely disturbed by a cortical lesion, whilst some other form escaped entirely; and yet in another case this relation may be completely reversed.

Many attempts have been made to determine the topographical localization of the sensory activities of the cortex, but they have all failed, because the nature of its functions was unknown. So long as investigators continued to think of the sensory cortex as the seat of processes underlying the full appreciation of touch, pain, heat, and cold, they were bound to find the clinical facts inexplicable. But as soon as it was evident that crude qualitative appreciation of these modes of sensation was due to the activity of the optic thalamus, it became possible to study the phenomena of sensory loss with some hope of discovering the part played by the cortex. Once we had determined that the physiological activity of the cortex was responsible for the recognition of spacial relations, intensity and relativity, it became possible to group the physiological processes which underlie sensation. But, before we can formulate any coherent conception of these cortical activities, it will be necessary to examine in detail the manner in which they are affected, as shown by the various tests belonging to the same order of sensation.

§ 2.—*Spacial Recognition.*

Some loss of power to recognize passive movements of the affected part forms one of the most frequent consequences of injury to the sensory cortex. In fact, it may be accepted for clinical purposes as a leading sign in the syndrome of cortical disease. It is, however, only one of the methods of discovering whether the spacial aspect of sensation is disturbed; the compass-test and "spot-finding," as we have employed them, are also explorations in spacial recognition.

But these three tests are not equally affected by a lesion of the cortex. The most delicate of them and the most susceptible of change is the power of appreciating a measured movement made passively. Next in order comes discrimination of the two compass-points applied strictly simultaneously.[1] Finally, topical localization or "spot-finding" is affected to the smallest degree of all the three spacial tests; for not only is it scientifically less precise, but the problem it presents to the patient is easier than with either of the other methods of research.[2]

This graduated relation between the disturbance of sensibility revealed by the three spacial tests, is evident from whatever point of view the records are examined. Thus, if the injury is a slight one, the only loss may be in the power of appreciating passive movement. A somewhat more severe disturbance affects discrimination of the compass-points, and finally "spot-finding" may also be defective.

The same law is manifest if we judge, not by the frequency with which any one of these three aspects of spacial recognition is affected over a series of cases, but by the number of digits which show disordered sensibility to each test in turn. Three-dimensional loss (appreciation of measured movement) occupies the largest number of fingers, two-dimensional loss is found to extend either to the same or to a smaller number of digits, and localization is disturbed over a still less extensive area.

I have summarized in the following table the records of twelve cases where spacial recognition was not uniformly disturbed over the whole hand. The numerals correspond to the number of digits found to be affected to each of the three tests. It is at once obvious that the widest loss of sensibility is shown by the range of passive movement necessary to excite appreciation, whilst the power of discovering the position of the stimulated spot is least extensively affected.

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[1] The compass-test cannot be used to measure the power of discriminating two contacts in cases where tactile sensibility is grossly disturbed. It presupposes that the patient is capable of recognizing touches with some approach to uniformity. Throughout this chapter I have carefully excluded this source of error, but have postponed its more detailed consideration to p. 126.

[2] Our method of testing the power of topical localization also depends somewhat on the condition of tactile sensibility. This association has been fully investigated and described by Graham Brown and Stewart [1], and is considered on p. 129.

Case	Measured movement	Compass-test	Localization
No. 10	5	4 + 1 introspective	3
No. 22	5	4 + 1 introspective	2
No. 4	5	4 + 1 introspective	2
No. 20	5	3	1
No. 19	5	2 + 1 introspective	1 + 1 introspective
No. 3	4 + 1 introspective	2	2
No. 7	5	1 + 1 slightly	0
No. 1	5	2	0
No. 17	4	3	1
No. 2	4	1	0
No. 12	3	2	0
No. 14	2	2	0

This graduated relation between the amount and the extent of the loss discovered by the three spacial tests comes out clearly in the following example (No. 3). Fortunately, tactile sensibility was not affected in this case, and the readings are therefore free from this troublesome source of fallacy.

	Measured movement	Compass-test	Localization
Thumb ..	1°, but direction not always accurate	Perfect at 1 cm.	Perfect
Index finger..	Between 2° to 3°, but slow	,, 1 ,,	,,
Middle finger	2° to 3°	,, 1 ,,	,,
Ring finger ..	Gross loss	Gross loss at 5 cm.	Slightly affected
Little finger..	Gross loss. Cannot even recognize posture of finger	All called "one" at 5 cm.	Definite, but slight loss

Here three-dimensional loss was so great over the little finger that the patient could not even recognize its position, when it had been moved passively into the extremes of flexion and extension. In the ring finger the disturbance was considerable but not so complete, whilst in the middle and index fingers a movement of from 2° to 3° was recognized slowly. The range of movement actually appreciated in the two thumbs was the same, but the answers from the affected hand were less prompt and the direction was not always indicated correctly.

The compass-test was affected over the ring and little fingers only, but over the latter the loss of discrimination was absolute when the points were 5 cm. apart.

Topical localization was slightly disturbed on the ring finger and the patient was less certain in his answers, but over the little finger the change was more definite. Out of five observations, he made two mistakes and at the same time complained that the test was more difficult than elsewhere over the hand.

Here then, both the degree of sensory change and the extent of the loss, measured by the number of digits affected, obeyed the law laid down at the beginning of this section. The earliest disturbance of sensibility is revealed by a test in three-dimensional recognition, whilst "spot-finding" is both last and least affected.

So far, I have spoken of the extent to which recognition in space was disturbed as if it was determined by measurement only. This is, however, not the case. For the first sign that any one of these varieties of spacial recognition is affected is the statement of the patient himself, that the tests are less easy on the abnormal digit. Even though the compass-test may give a threshold within normal limits, he is slower in his answers and finds greater difficulty in making up his mind as to whether he has been touched by one or two points. A similar state of uncertainty may be the first sign that localization is disturbed, although finally he may indicate correctly the spot touched. In all these instances, the change would be recorded as an "introspective" one. With intelligent patients, this introspective difference between similar parts of the two hands is a most valuable indication that a change in sensibility exists, even though it is too slight to become apparent by our somewhat coarse methods of measurement.

A more advanced affection is shown by a raising of the threshold; there may be no difficulty in obtaining perfect appreciation of passive movement and of the compass-test, but the angle through which the affected part must be moved may be five to ten times greater than normal, and the two points have to be separated to a distance far in excess of that necessary on the unaffected hand. In such cases correct recognition and discrimination are possible, provided the task is made easier.

The next stage of dissolution is shown by the absence of a threshold; movements to the extreme distance possible in any direction may not be recognized. The records are marred by uncertainty, guessing and hallucinations. The patient may describe movements, indicating their direction, although the part has remained rigidly in the same position; contact stimuli are particularly liable to be translated into sensations of movement. In the same way, the maximum separation of the compass-points possible over the affected part fails to lead to correct discrimination and the records may show uncertainty and guessing, even when one point only has been applied to the skin. An analogous tendency appears when the power of localization has been profoundly disturbed; the patient not only indicates erroneously the spot touched, but may give up all attempt to determine its position, saying, "I have no idea where you are touching me."

These various grades of defective recognition are well shown in the following case (No. 2), where all the fingers were in a different condition :—

	Measured movement	Compass test	Localization
Thumb	Normal (1°)	Threshold 1 cm.	Perfect
Index finger	Threshold raised (3° to 6°)	,, 1 ,,	,,
Middle finger	,, ,, (6° to 10°)	,, 1 ,,	,,
Ring finger	,, ,, (5° to 8°)	,, 1 ,,	,,
Little finger	Gross loss of threshold and confusion	Threshold 1·5 cm., with some uncertainty of response	,,

Here there was no difference between the sensory condition of the two thumbs; it was as easy for him to give correct answers to the three tests on the affected as on the normal hand. The first change appeared when he attempted to describe passive movements of the index finger; his answers were slower and a range of from 3° to 6° was required before he could appreciate the direction correctly. With the middle and ring fingers, the movement had to be made through an angle of from 6° to 10°, whilst in the little finger the loss was even greater. For he not only required movements of from 15° to 20° before he could recognize that some change of posture had occurred, but its direction was sometimes wrongly interpreted even at 25°.

In spite of this extensive disturbance in the recognition of passive movement, the only change to the compass-test was found on the little finger of the affected hand. Not only was it necessary to separate the two points to a distance of 1·5 cm., but he gave the following account of his sensations in this digit. "The thing that puzzles me on the little finger is that it seems like an echo. I don't know whether it is two, or one with an echo. When they are close together, I can't tell whether it is one point, which influences the part around, or two points. When they are wide apart, the second point falls outside this part influenced, and I know it is two."

Even when we consider the state of any one digit, we can frequently discover a similar graduated relation between the extent to which the three spacial tests are affected. Take for example the sensory records from the little finger in No. 2. Recognition of passive movement showed the profoundest disturbance; it was impossible to obtain a definite threshold, even with a wide range of alteration in posture, and the patient's answers were confused. Yet the compass-test revealed a threshold, with the points separated to a distance of 1·5 cm., and localization was entirely unaffected.

Whenever, therefore, spacial recognition is diminished as the result of a cortical lesion, appreciation of passive movement will be disturbed. Moreover, in any such case, this aspect of sensation shows the most extensive and also the most intense changes.

Next in order comes two-dimensional discrimination, as shown by the power of recognizing the two compass-points applied simultaneously.

Localization, or the capacity to determine the position of the stimulated spot, is affected less often, less extensively, and to a smaller degree than any of the other spacial tests.

This is an excellent example of the law, laid down by Hughlings Jackson, that the higher and most specific aspect of any function is affected before the lower. Sensory disturbances associated with lesions of the cerebral cortex are psychical manifestations, rather than the result of mechanical interference with conduction of afferent impulses. The physiological activities, which directly underlie sensation, are struck in the centre of their production. We have no longer to deal, as on lower levels, with loss of sensation caused by the cutting off of afferent impulses from a normal receptive centre. All such expressions as "joint-sense," "proprioceptive impulses," "epicritic and deep sensibility," adequate terms at certain other sensory levels, cease entirely to have validity when we are considering the disturbances in spacial recognition associated with a cortical lesion.

Such a lesion, when it interferes with spacial recognition, first disturbs that function which is psychically most difficult, and then in order those of less complexity. We are so near the psychical act of sensation that we must now classify disturbances of sensibility under categories we can recognize introspectively, and we are not compelled to group them according to their local origin, phylogenetic qualities, or physiological history, as was the case with lesions at lower levels of the nervous system.

§ 3.—*Intensity.*

Touch, pain, heat, and cold form a natural group with certain features in common. Each of these aspects of sensation depends on the action of measurable physical stimuli; the impulses generated are specific and, on the physiological levels of the nervous system, they are gathered together into separate afferent streams, which can be interrupted independently of one another.

But a cortical lesion never completely abolishes sensibility to touch, pain, heat, or cold. Even when the whole sensory cortex is thrown out of action, contact, especially of an object moving over hair-clad parts, may produce a definite tactile response. Under such conditions, contact-stimuli produce a vague sensation of "something happening" within the body, without immediate appreciation of variations in intensity. In the same way, heat and cold may be correctly discriminated, although the patient cannot recognize differences in the degree of thermal stimulation. The sensation evoked by a prick may not seem so "sharp" or so "clear" as over normal parts, but the discomfort it produces may be greatly increased.

The power of recognizing contact, especially of a moving object, and appreciation of the qualitative aspects of thermal and painful stimuli, are functions of the optic thalamus; the activity of the cortex endows us with the capacity to estimate the relative intensity of such stimuli. It is obvious, therefore, that all these forms of sensibility must be affected in a similar way by a cortical lesion. Their qualitative appreciation is already provided for elsewhere; but the clarity and definiteness of the sensation and the stability of the response to repeated stimuli are a function of cortical activity.

Now, from the cortical aspect our tests for measuring sensibility to touch, temperature, and pain are not of equivalent value. The tactile hairs demand a high degree of constancy and graduated response to stimulation; this test, therefore, shows the greatest change amongst the group we are now considering.

Appreciation of heat and cold, as we are forced to examine it clinically, depends comparatively little on cortical activity. For, whilst it is easy to say whether an object is hot or cold, it requires an intelligent patient and unusually favourable conditions to test the relation of two warm stimuli to one another, or to determine the threshold for heat and cold with approximate accuracy.

These difficulties are multiplied a hundredfold in the case of pain. A prick is the only effective means of applying a measurable painful stimulus to the skin, and any sharp object, such as a needle, produces first of all a recognition that the stimulus is pointed; further pressure adds to this a sensation of pain. It is almost impossible to prevent the patient from saying that he is pricked, whenever he appreciates that the stimulus is a sharp point, and, if he is told to wait until he obtains a distinct sensation of pain, we run the risk of placing the threshold too high. In the vast majority of cortical disturbances, the threshold for prick is the same on the two sides. But in rare instances it would seem as if the threshold were raised somewhat over the affected parts, and the patient complains that the "feeling" is different. How far this is due to a profound change, not uncommonly present, in the sensation produced by the point apart from pain, and how far it may be caused by a slight raising of the pain-threshold, is dealt with in Chapter VI [p. 140]. Whatever the true significance of this phenomenon, appreciation of a prick is affected less often than any other form of sensation.

Whenever the power to appreciate touch, pain, heat, or cold is disturbed, in consequence of a cortical lesion, measurements with hairs of graduated strength invariably reveal some loss of tactile sensibility. Out of eleven cases, where one or more of these qualities of sensation showed some defect on the hand, tactile appreciation was disturbed in all; five showed some loss of thermal sensibility, whilst measured prick was defective in one case only.

If these eleven cases are gathered together to form a table, it will be seen not only that tactile sensibility is most often affected, but the loss is invariably greater in amount than that of either of the two other forms. Similarly the defects shown by the thermal tests are greater than those which appear in the records of measured prick.

Case	Tactile hairs	Thermal tests	Prick
No. 5	Gross loss	Affected	Threshold raised
No. 10	,, ,,	,,	Normal
No. 17	,, ,,	Introspective	,,
No. 4	,, ,,	,,	,,
No. 18	,, ,,	Slightly affected	,,
No. 14	Threshold raised	Normal	,,
No. 13	,, ,,	,,	,,
No. 20	,, ,,	,,	,,
No. 12	,, ,,	,,	,,
No. 1	Threshold slightly raised	,,	,,
No. 7	,, ,, ,,	,,	,,

A similar relation is evident, if we judge by the number of digits affected in each particular instance. The following table shows that the extent of the loss to graduated tactile stimuli exceeds, or is as great as, that of any other form of sensibility belonging to this group. Next in order comes the number of fingers which exhibit some lack of thermal appreciation, whilst in most cases prick is not affected anywhere.

Case	Tactile hairs	Thermal tests	Prick
No. 6	5	2 + 2 introspective	3
No. 10	5	3	0
No. 13	5 threshold slightly raised	0	0
No. 4	3 + 2 introspective	3 introspective	0
No. 20	1 + 4 „	0	0
No. 17	2	1 introspective	0
No. 14	2	0	0
No. 1	2	0	0
No. 12	1 + 1 introspective	0	0
No. 7	1 + 1 „	0	0

In all attempts to record the amount or the extent to which these forms of sensibility are affected we cannot depend entirely on measurement. Introspective differences, irregularity of response and hallucinations must all be carefully taken into account.

It is more particularly in this group of tests, dealing with response to stimuli of different intensity, that introspection is of the greatest value. The patient insists that the impression he receives is "less plain," "less certain," over affected parts; the threshold may be slightly raised only, or it may be impossible to say that there is any measurable difference between similar parts of the normal and affected hands, and yet he insists "the two hands are different." "It is harder for me on the affected hand; I can't be certain that I am right, but it's easy enough on the normal hand."

No. 2 was a splendid example of this degree of sensory affection. For, as far as touch, temperature and pain were concerned, the little finger showed a disturbance of sensibility which, though not apparent to our rough tests, was obvious to the patient. He gave a perfect series of answers on the right and left little fingers even with a hair of 14 grm./mm.^{2}, but the response was slower from the right than from the left. He said, "The first two or three touches were fairly clear; then it seemed to become more difficult and I wasn't so certain. I was doing it with an effort on the affected hand (right) instead of not thinking about it." With the same hair on the two thumbs he said, "That is quite easy; there is no effort on either side."

The threshold for the appreciation of heat and cold did not differ materially on the two little fingers; but he asserted that the impression he received was not the same. "There is a difference in the left hand (normal); it is immediate, whilst in the right it seems to change gradually. The ultimate sensation is the same, but it is quicker in the left hand."

On testing this patient with measured pricks, it was difficult to be certain whether the threshold was raised or not; but he never doubted for a moment that he received a different impression from the two little fingers, whilst the sensation evoked from the two ring fingers by the same stimulus was identical. If the algesimeter was set at 3·5 grm., it was said to "prick" on both little fingers; on the affected hand, however, it was "not so plain" as on the normal one.

Here, then, is an instance where, as far as the intensity of tactile, thermal and painful stimuli is concerned, no measurable abnormality could be discovered: and yet, in every case, the patient was certain that the sensation from the two hands was different.

When sensation is more gravely disturbed, the threshold rises over the affected part; it may still be possible to obtain a perfect series of answers, but a stronger stimulus is required. The normal hand may respond perfectly to a hair of 14 grm./mm.^{2}, whilst imperfect records may be obtained on a similar part of the other hand, until we employ a hair of considerably greater strength. In such cases, the patient is usually conscious that the sensation evoked is different, and hallucinations are liable to appear, especially if the testing has continued for an unduly long period. In the same way, although crude recognition of heat and cold may show no confusion, the patient may not be able to appreciate heat below about 35° C., or cold above about 26° C., whereas on normal parts the thresholds are respectively about 30° C. and 28° C.

In many cases, a further stage is reached in sensory dissolution. No threshold can then be obtained by increasing the stimulus within certain limits. Normally a hair of 14 grm./mm.^{2}, or at any rate one of 21 grm./mm.^{2} evokes a perfect series of answers from the palmar aspects of the tips of the fingers. When, however, we speak of the threshold as unobtainable, we mean that even a hair of 100 grm./mm.^{2} does not necessarily or materially increase the accuracy of response. It is not safe to employ hairs of greater bending strain, as they are liable to produce a sensation of pricking; even those between 70 or 80 grm./mm.^{2} may occasionally seem to prick, over the tips of the fingers.

With the disappearance of a threshold, hallucinations frequently mar the records; the patient responds when he has not been stimulated, and often insists that the hair has not been removed, but still presses on the surface. At other times, it seems to him, as if the hair was being dragged along the skin, instead of being applied to one spot and then removed. This translation of contact into a sensation of movement is one of the most striking consequences of some disturbance in the sensory cortex.

Case 5 was an excellent example of such changes in the appreciation of touch, heat, cold and pain.

	Tactile hairs	Thermal tests	Prick
Index finger ..	Threshold destroyed; 23 and 100 grm./mm.^{2} gave the same number of correct answers. Irregular response. Contact thought to be movement across the surface	No certain appreciation between about 26° and 40° C.	8 grm. or more
Middle finger	Threshold raised 23 grm./mm.^{2} ..	Unable to distinguish 35° and 40° C.	8 grm.
Ring finger ..	,, ,, 23 ,, ..	Threshold for cold 27° C., for heat 34° C.	8 ,,
Little finger ..	Much introspective difference; threshold slightly raised	Introspective change only	4 grm.; same as normal little finger

On the normal hand, the threshold for measured touch was in every case below 21 grm./mm.^{2}; that is to say, that with a hair of this bending strain, a uniform series of replies could be obtained from all his digits. With an external temperature of 18° C., it was found that the threshold for heat, on the fingers of the normal hand, was about 30° C., that for cold was about 27° C. He had no difficulty in recognizing the difference between a silver tube containing water at 35° C., and one at 40° C., presented in different order and irregular sequence. Prick was distinctly appreciated on all the normal fingers, when our spring algesimeter registered 4 grm.

Let us examine more in detail the condition of the index finger of the affected hand, as compared with that of the normal side.

A series of observations with the tactile hairs, carried out in the usual way, yielded the following readings. Each unit represents appreciation of a contact of measured force indicated in grm./mm.^{2}, whilst a nought signified that the touch was not perceived. It is evident that no true threshold could be obtained to touches of this order, for the number of correct answers with a hair of 23 grm./mm.^{2} is the same as that to a hair of more than four times the bending strain.

	R. (affected)	L. (normal)
21 grm./mm.^{2}	—	iiiiiiiiiiiiiiii
21 grm./mm.^{2}	oooooooooooooooo	
23 grm./mm.^{2}	iooiooiooooooiioo	
35 grm./mm.^{2}	ioiioiooiooooioi	
70 grm./mm.^{2}	iiiiiiooiiiiiooo	
100 grm./mm.^{2}	ooiooiiioooiioooo	
23 grm./mm.^{2}	ooooooooioiooooi	
21 grm./mm.^{2}	—	iiiiiiiiiiiiiiii

This bald record, however, gives an incomplete idea of the sensory disturbance. The patient said, "On the left (normal) side I felt as if you were putting on and taking off a fine wire point. On the right hand, it was exactly as if a mosquito drew its leg along the surface. I never felt a distinct pressing on. Sometimes I felt it vaguely as if it was still there, and I wanted you to press it and take it away again, so that I might be certain if it was there or not."

On the affected index finger, he could not appreciate heat or cold with certainty between about 26° C. and 40° C., whereas on a bright spring-like day, the temperature of the room being 18° C., the indeterminate zone lay between about 26° C. and 30° C. over the normal hand.

This extension of the indeterminate zone on the affected hand is one of the characteristic features of thermal sensibility, when it is disturbed from a lesion of the cortex. An even more striking defect is inability to distinguish two stimuli of the same thermal quality but of different intensity. I have seldom examined a more complete example of this defect.

Index finger	R. (affected)	L. (normal)
Compared—		
35° and 40° C.	"No difference"	"The second was much hotter"
40° and 35° C.	"No difference"	"The first was hotter"
35° and 40° C.	"No difference"	"The second was hotter"
35° and 45° C.	"Both hot; the last hotter"	"The last was hotter"
45° and 35° C.	"No difference"	"The first was hotter"
35° and 45° C.	"No difference"	"The last was hotter"
45° and 35° C.	"First hotter; the second was cold, or nearly so"	"The first was hotter"
25° and 20° C.	"Cold; no difference"	"Neutral and cold"
20° and 25° C.	"The same; just about cold"	"Cold and neutral"
25° and 20° C.	"The same; they are not neutral"	"On the cold side and distinctly cold"
20° and 25° C.	"Both are ice cold"	"First much colder; the second just on the cold side of neutral"

When tested with the warm and cold tubes, he said: "There is a great difference between the two fingers; the right never changes its mind, whereas with the left (normal) the temperature seems to grow upon it; with hot I feel it growing hotter, until it reaches a certain stage and then stops. Cold does the same thing. I was baffled on the right hand; I don't know how to describe it. It sometimes seemed ice-cold and sometimes nothing at all; but there was never any difference between the two tubes." He was first tested with the cold tubes on the affected hand, and as soon as the change was made to the normal hand, he burst into laughter saying, "I could not have believed that the difference could be so great. Here I never had a moment's doubt; the first was just off tepid and the second was colder still, or vice versa as the case might be."

To prick, the threshold was decidedly raised and although 4 grm. was sufficient on the normal index finger to evoke the sensation of pricking, 8 grm. or more was required on the right hand. Moreover, he said, "I feel it more on the left hand; it is quite soft on the right."

Thus, we can sum up the results reached in this section as follows:—

(1) Sensibility to touch, heat, cold and pain is never abolished as the result of a true cortical lesion, provided the period of shock has passed off and epileptiform attacks are absent.

(2) Sensibility may, however, be profoundly modified. This change shows itself first in the patient's introspective recognition that the sensation obtained from the normal and affected parts is not similar Next, this difference may become manifest in a raised threshold. A still graver disturbance is shown by inability to obtain a threshold within reasonable limits; such records are liable to be disturbed by hallucinations and other irregularities of response. At this stage of dissolution, the patient may not be able to appreciate with certainty temperatures between about 26° C. and 40° C., nor to discriminate between two hot or two cold stimuli, easily recognizable on the normal hand.

(3) Whenever the graduated tests for touch, heat, cold or prick demonstrate that sensibility is changed, the tactile hairs always discover the gravest disturbance, heat and cold are less and prick least affected. By this I mean, not only that the loss is more intense over any one part, but also that it extends to a larger number of digits on the hand.

(4) This is probably due in part to the more delicate and complex appreciation required by the test of the tactile hairs, compared with the crudity of those we can apply clinically for heat, cold and especially for prick. But it also depends on the relative extent to which these stimuli appeal to the activity of the optic thalamus. A prick, as far as its painful element is concerned, depends for recognition almost entirely on this organ, whilst the "pointedness," "clearness," "sharpness" are the result of cortical activity. On the other hand, the graduated hairs require for their appreciation a reaction to different intensities; this reaction must be constant and correspond relatively to the strength of stimulation. When once the maximum threshold has been exceeded,

the answers must be free from gross irregularities. All these factors in contact-sensibility depend on the sensory cortex and are therefore mos disturbed in consequence of such lesions as I have dealt with in this paper.

(5) All these changes in sensibility can be grouped together under the heading of defective appreciation of intensity; for, at bottom, they depend on an inconstant reaction to graduated stimuli. This tends to produce that characteristic confusion and want of definition, which are manifest throughout the records of touch, pain, heat and cold in many cases of cortical injury.

§ 4.—*Recognition of Similarity and Difference.*

Appreciation of the shape, relative size, and nature of common objects is frequently disturbed in cases of cortical lesion, and has been erected into one function under the name "stereognosis." But this term is a bad one, because it applies strictly to recognition of form only; it cannot possibly be made to include the relative appreciation of weights, the most easily measurable member of this group of tests. Nor can it comprise the many other means of examining the patient's power of recognizing the similarity or difference of two objects placed in the hand, all of which may be more or less simultaneously affected by a lesion of the cerebral cortex.

When we were dealing with the consequences of destruction at subcortical levels of the nervous system, the success or failure of all the tests of this group depended on more than one stream of impulses; the patient might fail if the test was carried out in one way, but make a good record by another procedure. Thus, when the interference with conduction is subcortical, the power of appreciating differences in weight depends on two separate streams of afferent impulses, one concerned with tactile impressions, and the other with recognition of passive movements. If the hand is fully supported, addition and removal of weights can be correctly appreciated only when tactile sensibility is perfect; subcortically, this test is another method of discovering whether touch is or is not affected. When, however, the hand moves freely in space, and the patient is told to "weigh" the objects placed upon the palm, his power of estimating weight depends on the integrity of spacial recognition. It is, in fact, another indirect test for appreciation of movement.

As soon, however, as we come to deal with disturbances of cortical origin, we are face to face, not with an interference with conduction to an intact centre, but with destruction of the activities of that centre itself. The power of estimating weights is now a primary, and not a secondary function; and, if it is disturbed by a cortical lesion, all aspects of the tests are more or less affected.

The power of appreciating differences in weight is no longer linked up directly with postural recognition, or with tactile sensibility. These functions may be entirely dissociated. Thus, in No. 18, measured movement was in no way defective, and tactile sensibility was disturbed to a scarcely perceptible degree, and yet the power of appreciating differences of weight was gravely affected. Conversely, in No. 13, the patient was able to discriminate differences in weight, and yet recognition of movement and tactile sensibility were considerably disturbed.

Again, it may happen that perfect appreciation of graduated contacts is accompanied by loss of power to estimate weight. In two cases (No. 16 and No. 21), where this condition was present over the whole of the affected hand, it was associated with inability to recognize passive movement in all five digits. At first sight it might be supposed that the two lost functions were bound up together, were it not for another group of cases [Nos. 13, 19, 22], where the records of measured movement showed the grossest defects, but the relation between two weights could be correctly appreciated.

Thus it is evident that the power to recognize differences of weight is a function of the cortex, independent of tactile sensibility, or of the appreciation of measured movement and posture of the affected parts. At all levels of the nervous system below the cortex, discrimination of weights depends on the manner in which the test is applied. If tactile sensibility is diminished, the patient is no longer able to recognize the relation of two weights placed on the hand when fully supported, whereas a proper appreciation of movement is required for the due recognition of weights laid on a hand that is moving freely in space. But, from the point of view of cortical activity, discrimination of weight is a function independent of either postural or tactile recognition.

The faculty of recognizing the shape of objects placed in the hand is a closely allied function, as far as the sensory activities of the cortex are concerned. But, whereas graduated weights can be applied to each separate finger, it is impossible to use the tests for appreciating shape in this way. In order to prove that these tests fall into the same sensory group as those for weight, from the point of view of cortical activity, we are compelled to fall back upon cases where, at any rate, a considerable part of the palm is affected as well as the digits. The patient is then allowed to roll the solid objects of different shape between the fingers and the palm.

On running through the records of my series of cases, it is at once apparent that recognition of form in three-dimensions rises and falls *pari passu* with the power of appreciating the relation of two weights; when the one aspect of sensory discrimination is disturbed, the other is also affected. Thus, No. 18 had lost all power to recognize the shape of solid objects rolled between his thumb, index, and palm, and could not recognize addition or subtraction of weights, although appreciation of passive movement was not affected, and the tactile threshold was but slightly raised. Conversely, in No. 14, recognition of form and weight was perfect, although the power of appreciating measured movement and the tactile hairs was obviously defective.

Throughout this research, I have used wooden figures cut to scale, and of six different geometrical shapes. Their size was such that they could be easily recognized in the normal hand, if the patient were sensitive and intelligent, even when rolled over the palmar aspect of the fingers.

It is unfortunately impossible, clinically, to use several sets of solid figures of different sizes in order to test appreciation of form; for the time required to make so long a series of observations would be too great, and in most cases they would end in confusion on the part of the patient; this must be avoided even at the cost of reducing the measurable aspect of the test. Should this faculty of recognizing the form of objects be diminished, but not entirely lost, our method does not enable us to work out a threshold, as was the case with weights; the results are purely relative, and cannot be expressed numerically. But when the disturbance is extreme, the patient not only says he has no idea of the form of the geometrical figures we habitually use, but he may also fail to recognize the shape and nature of common objects such as a knife, coin, or pencil placed in his hand. This is the well-known condition usually called "astereognosis."

The power of recognizing stuffs by their difference in texture also belongs primarily to this group; it is closely associated with ability to appreciate differences in weight and form. But this aspect of the test presupposes perfect tactile sensibility. If touch is vague and uncertain, it is obvious that the patient will not be able to recognize the characteristics of the stuff beneath his fingers as readily as with the normal hand, and so will fall into mistakes that have nothing to do with the power of appreciating similarity or difference. The relation of this test to those for the recognition of weight and form can only be established in cases where tactile sensibility is perfect.

Fortunately I have examined one such instance of this dissociation in an extreme form (No. 16). Touch was nowhere disturbed, but the patient was unable to distinguish velvet, silk, flannel or cotton anywhere over the affected hand. He had also lost the power of recognizing differences in weight, whether the hand was supported or free to move, and had no idea of the shape even of common objects placed in the palm. Thus, provided tactile sensibility is normal, it is possible to show that the recognition of differences in weight, form and texture is the expression of one aspect of cortical activity.

A striking example of this law is seen in the case of No. 3, where all the digits were not in the same condition (*vide* table). Tactile sensibility was everywhere normal, but the three tests, weight, form and texture, showed considerable loss over the little, ring and middle fingers. Appreciation of measured movement was disturbed to a greater or less extent over the whole hand.

	Weight	Texture	Form	Tactile hairs	Measured movement
Thumb	Perfect	Perfect	..	Perfect	Introspective (1° perfectly)
Index	,,	,,	Normal	,,	Affected (2°—8°)
Middle	Defective	Defective	,,	,,	,, (2°—8°)
Ring	Gross loss	Gross loss	Complete loss	,,	,, (8° badly)
Little	,, ,,	,, ,,	,, ,,	,,	,, (not under 5°)

The tests we have considered in this section show that the power of discriminating differences in external objects, apart from the intensity or spacial attributes of the sensations they evoke, is a fundamental function. It is not secondary to the due appreciation of some other sensory quality, such as touch or spacial projection. This is strictly true of the discrimination of weight and form, but full recognition of differences of texture is only possible, when tactile sensibility is also unaffected.

All these tests, the weights, the different shapes in three dimensions and stuffs of various textures, demand the same power of discrimination. Some offer a more difficult task than others, but they do not stand to one another in a graduated scale of difficulty like the three tests for spacial recognition, or the tactile, thermal and painful stimuli we have employed in this work. In cases where they can all be employed in consequence of the whole hand being affected, they are usually found to fail together. Tests capable of measurement, such as those for weight, obviously reveal a slighter degree of change than those which consist in the recognition of certain fixed shapes or textures; for, in this latter group, the patient has simply to appreciate the nature of an object whose character cannot be varied.

In conclusion, there can be little doubt that the faculty of recognizing the similarity and differences of objects brought into contact with the body depends upon one of the fundamental activities of the sensory cortex. It can be disturbed apart from the power of appreciating spacial relations and independently of the reaction to stimuli of varied intensity, or it may be preserved, when these two functions are grossly defective.

§ 5.—*Analysis of the Tests which depend upon more than one of these three Cortical Activities.*

So far, I have treated each of these sensory tests as if it made a specific appeal to one of the three aspects of cortical activity. This is manifestly an incomplete statement of the case. For, if the physiological workings of the cortex endow sensibility with relations in space, intensity, and individual character, it is obvious that these functions will be exercised on all afferent materials presented to its action.

There are but few tests which do not depend in some way on the appreciation of contact, and wherever tactility comes into action the question of its spacial relations, intensity or characteristics must of necessity arise. But, in the preceding sections of this chapter, I have attempted so to select my instances that the particular test in question shall give, as far as possible, an uncomplicated answer. Thus, when discussing spacial sensibility, I have chosen cases where the power of appreciating graduated intensity was little affected. In the same way, the capacity to recognize various stuffs by their texture was treated, as if the condition of tactile sensibility played no part in the test.

It was necessary to adopt this deliberately partial standpoint in order to elucidate the nature of the sensory activity of the cortex. For it is obvious that the simultaneous compass-test depends on the integrity of tactile sensibility; at lower levels of the nervous system it is often impossible to apply this test, because the patient cannot appreciate any tactile stimulus, however intense. Even with a less complete interference, the impression made by contact may be so feeble that the patient does not receive the afferent materials necessary for discrimination. Under such conditions the compass-records would show little more than the effect of the loss of touch. But we have many better methods for measuring defects in tactile sensibility, and the true value of the compass-test lies in the fact that it is the best means at our disposal for exploring two-dimensional discrimination

By careful selection of cases it is possible to find specific instances where each of the tests we have used exhibits the changes in one only of the cortical activities on which it depends. But, from the point of view of the general facts presented by cerebral lesions, such exclusion is wasteful and inconvenient. It behoves us, therefore, to discover, if possible, some peculiar feature of the records of each of these tests which will reveal the particular activity at fault. In many cases two or even three of these activities may be defective; here the records will show the profoundest changes, but they are useless for that analysis upon which we are intent in the present section.

In a recent paper, Graham Brown and Stewart [1] put forward their views on disturbances of localization and discrimination in cases of cerebral lesions; their results were based on a long series of observations mainly on one patient, but the conclusions to which they arrived are of the greatest interest. They believe that a sensation of touch has three attributes—its character, individuality and position. Every sensory effect produced by a touch must evoke a state different from that conditioned by all other tactile stimuli; this is its "character." But each sensation of touch must resemble all those arising from stimulation of the same spot, and this is what they call its "individuality." But, apart from this individual similarity to others that have preceded it, a touch has definite "position." The two authors then analyse along these lines their experiments on topical localization and discrimination of the compass-points.

My observations are not extensive enough, and the tests I have used are relatively too coarse, to permit any criticism of their results. My method of attacking the problem differs from that by which they reached their results. I have attempted, by ranging widely over a number of suitably selected cases, to discover conditions where the various factors in cortical activity were dissociated; thus I have been able to observe the behaviour of the patient under simultaneous application of the compass-points, when other spacial tests were affected, but tactile sensibility was perfect. Conversely, these results were compared with the compass-records obtained when spacial recognition was normal, but there was grave want of response to tests of graduated intensity. In this way, certain general rules have emerged, which I shall proceed to consider more in detail under the heading of each separate test.

(*a*) *The compass-test.*—This test above all others is liable to be disturbed by inequalities of tactile appreciation. It is essentially a measure of the power of discriminating two points applied simultaneously. If, however, the two contacts evoke sensations of different and inconstant intensity, in spite of the most careful application, the patient will have difficulty in making up his mind as to whether he has been touched with one or two points, and yet from other tests it may be unlikely that the spacial aspect of sensation is disturbed.

From the records of selected instances, where in the one case spacial recognition was affected, but tactile sensibility was perfect, and in the other the condition was reversed, I think it is possible to enunciate the following general rule. So long as the power of reacting uniformly to touches of graduated intensity is not impaired, defects in two-dimensional discrimination show themselves in a tendency to speak of both single and double contacts with the compass-points as "one." Thus, according to the method we use for recording the results of this test, errors will be plentiful in the lower line, because many "twos" are called "one," whilst the upper line will be almost clear, because there is an habitual tendency to call all contacts "one."

On the contrary, if the appreciation of tactile stimuli is inconstant, as shown by the answers obtained to the tactile hairs, the compass-records will have a different appearance, provided spacial recognition is not affected. Just as many errors may appear in the upper as in the lower line; one contact is called "two," and two are said to be "one" with almost equal frequency. Moreover, a considerable increase in the distance between the points may make little difference in the number and proportion of these mistaken answers.

Let us first of all consider the case where the appreciation of tactile intensity was not disturbed. No. 3 is a good example of such a condition. The left hand was alone affected and all five digits showed some want of recognition of passive movement. Tactile sensibility was, however, remarkably good; a hair of 21 grm./mm.^{2} gave perfect records over the palmar aspect of all the terminal phalanges and the patient could discover no difference in the sensation evoked from the two hands. It was obvious, therefore, that if discrimination of the compass-points was defective the disturbance must have been mainly due to want of spacial recognition in two dimensions. The compass-records were perfect over the thumb, index, and middle fingers; but over both ring and little fingers every application, whether of one or two points, was called "one," even when they were separated by a distance of 5 cm.

Moreover, the patient added, "I feel the pressure but I can't be certain that it is two. It doesn't feel like it does on the other fingers."

The progressively increasing disturbance of the compass-records over the little, ring and middle fingers in No. 22 is another striking example of the loss of two-dimensional discrimination. All the digits reacted constantly to a hair of 21 grm./mm.^{2}, as on the normal hand; the only change on the middle and ring fingers was an introspective one, and even this abnormality was absent from the little finger. Here the two hands reacted alike to a hair of this bending strain. The records of the compass-test worked out as follows on the three fingers:—

Little finger—			
0·5 cm.	1	10 Right.	0 Wrong
	2	7 Right.	3 Wrong
1 cm.	1	10 Right.	0 Wrong
	2	10 Right.	0 Wrong
Ring finger—			
2 cm.	All called "one"		
3 cm.	1	10 Right.	0 Wrong
	2	7 Right.	3 Wrong
4 cm.	1	10 Right.	0 Wrong
	2	10 Right.	0 Wrong
Middle finger—			
3 cm.	All called "one"		
4 cm.	1	10 Right.	0 Wrong
	2	2 Right.	8 Wrong
5 cm.	1	10 Right.	0 Wrong
	2	3 Right.	7 Wrong

Here we see a steadily increasing tendency to call all the contacts "one" at wider distances, as we pass from finger to finger towards the centre of the disturbance.

The account obtained from intelligent patients confirms the conclusions drawn from examining the records. They complain that they cannot feel the difference between one and two points. "I don't feel two as I do on the other fingers," said one of them in whom tactile sensibility was little, if at all, disturbed. "I have to stop and think how big the feeling is; sometimes it seems over a larger area, and then I say 'two.' I don't really know at all." This is the secret of this form of change; it is an "agnosia" of greater or less intensity.

When, on the contrary, the records of the compass-test are disturbed by a defective and inconstant reaction to graduated tactile stimuli, the records exhibit a constant confusion. As many single touches may be said to be "two" as double contacts are recorded as "one." For example, in No. 18, passive movements were perfectly appreciated in all the digits, but the compass-test yielded defective records over the thumb, index, and middle fingers. This coincided exactly with the extent of the disturbance of tactile sensibility; for, over the thumb, he did not give a perfect series of answers with any hairs of less bending strain than 35 grm./mm.², whilst over the index finger even 100 grm./mm.² yielded a series in which he failed to appreciate six out of sixteen consecutive contacts. Over the middle finger, the condition was somewhat better, and he responded to 23 grm./mm.², which is still considerably above the pressure to which he reacted well on the similar digit of the normal hand.

Bearing in mind this defective reaction to contacts and the complete absence of any loss of spacial recognition, the following compass-records form a pronounced contrast to those given in detail above.

Thumb—			
1 cm.	All called "one"		
3 cm.	1	6 Right.	4 Wrong
	2	6 Right.	4 Wrong
Index finger—			
1 cm.	1	5 Right.	5 Wrong
	2	4 Right.	6 Wrong
3 cm.	1	4 Right.	6 Wrong
	2	8 Right.	2 Wrong
Middle finger—			
1 cm.	1	7 Right.	3 Wrong
	2	6 Right.	4 Wrong
3 cm.	1	6 Right.	4 Wrong
	2	6 Right.	4 Wrong
Ring finger—			
1 cm.	Perfect record		

No. 15 gave a striking demonstration of the same sort of defect. By a remarkable chance, a wound of the vertex had disturbed the power of recognizing passive movement in the left foot, and here also two-dimensional discrimination was somewhat defective. Touch was in every way normal. Conversely, over the right foot tactile sensibility was grossly affected, but passive movements were perfectly appreciated. The contrast between the compass-records on the two feet is most instructive.

Right Sole (tactile loss)			
3 cm.	1	6 Right.	4 Wrong
	2	5 Right.	5 Wrong
4 cm.	1	6 Right.	4 Wrong
	2	5 Right.	5 Wrong
6 cm.	1	7 Right.	3 Wrong
	2	4 Right.	6 Wrong
10 cm.	1	10 Right.	0 Wrong
	2	10 Right.	0 Wrong

Left Sole (spacial loss)			
3 cm.	1	10 Right.	0 Wrong
	2	6 Right.	4 Wrong
4 cm.	1	10 Right.	0 Wrong
	2	9 Right.	1 Wrong

The patient complained that on the right sole he found great difficulty, "because they all seem to run into one another. The touch seems to linger on here; it is difficult to find out, if it is the one you put on last, or if it is two together."

Here a slight defect in two-dimensional discrimination on the left foot led to some double contacts being mistaken for single ones, whilst on the right sole the tactile loss resulted in confusion, evident in the records both of one and of two touches.

(*b*) *Topical localization* ("spot-finding").—The method we have used for testing the power of one-dimensional localization is a comparatively coarse test, and allows a considerable margin of possible error. Moreover, the results cannot be expressed numerically. The patient is told that he will be touched over the various digits between the joints and that he is to indicate the exact spot on the hand of an assistant. The normal hand is first tested, so that he may understand what is required of him. This also enables us to judge of his natural aptitude and trustworthiness, before undertaking exploration of the affected parts. Any answer that lies over the correct phalanx is recorded as a successful response, and thus the margin of error may be nearly a centimetre distal or proximal to the spot actually touched.

Then, too, in contrast with the long series of experiments of Graham Brown and Stewart, I have as a rule made no more than six observations on any one finger, a number utterly insufficient for any conclusion, except that topical localization was or was not affected. Each digit was then carefully re-tested for the appreciation of two points applied consecutively, which has been shown elsewhere (Head and Holmes, [4]) to be a closely analogous function.

But, by selecting cases in which the various sensory activities of the cortex were dissociated, it has been possible to reach a definite conclusion. In this I have been greatly aided by the patient's own account of his difficulties.

In No. 3, the reaction to graduated tactile stimuli was perfect, but the three-dimensional aspect of sensation was disturbed in all five digits, reaching a maximum in the ring finger. I therefore chose the palmar aspect of the four post-axial digits for a more extended series of observations on topical localization. A tactile stimulus was planted on each phalanx in irregular order and the patient indicated on the hand of an assistant the position of the spot touched. Twelve contacts were made, first on the normal and then on the affected side; this was repeated three times on each hand to the number of seventy-two observations in all. Every touch on the normal hand was correctly localized, but the answers from the fingers of the affected side showed a defect increasing in the post-axial direction. Thus, whilst all nine stimuli were correctly localized over the left index, he made one mistake over the middle, two over the ring and six over the little finger. Of these six errors, one was placed on the wrong digit, three were more or less distal, and two were proximal to the spot stimulated. Two out of three mistakes on the middle and ring fingers consisted in choice of the wrong digit.

This inability to decide which digit had been touched was strikingly evident in No. 22. The maximum point of the sensory disturbance was the middle finger, but in the thumb, index, ring, and to a slighter degree the little finger, he had difficulty in appreciating passive movement. Now, whenever he was asked to localize a touch over the three middle digits, his answers were slow and hesitating. He moved them backwards and forwards as if he was playing the piano, and then indicated the spot on my assistant's hand, which he imagined I had touched on his own, lying hidden from his sight. He said, "I couldn't tell which of the three fingers it was accurately: but, when I moved them, then I could tell.". As a matter of fact, he was not always accurate, even in his selection of the finger that had been touched.

In another patient (No. 19), there was some loss of appreciation of passive movement in all five digits, reaching a maximum over the little finger. The tactile threshold was just perceptibly raised over the ring and little fingers; but the defect was extremely slight, and would not have been measurable, had he not been unusually sensitive over similar normal parts. But his power of localizing a touch was obviously defective. His answers were slow, and the tone of his voice gave a better idea of his state of doubt than the actual records. He said, "I have difficulty in making up my mind whether you are touching me on the little or ring finger." Out of eight contacts, two were wrongly placed, and, with two others, he said he did not know on which finger they lay, though he finally chose correctly. When the compasses were applied consecutively with the points separated by a distance of 0·5 cm., he gave one false and one doubtful answer out of ten double contacts on the little finger; on the ring finger, he found "it was a great effort to decide whether it was one or two," although he ultimately made no mistakes.

Thus it would seem that, when localization is defective, because the spacial element in contact sensibility is disturbed, the error takes the form of inability to decide on what finger, or on what part of it, the touch has fallen.

This is a great contrast to the condition discovered in the thumb of No. 9. I have chosen this digit, because here the power of recognizing passive movements was not affected, as it was in the index and middle fingers. The tactile threshold was raised, so that he did not give a perfect series of answers to any hair under 100 grm./mm.² He had no doubt which digit had been touched; but he passed his indicating finger up and down, saying, "It was somewhere on the thumb, I can't say where." The thumb, the index or the middle finger were at once named correctly as the site of the stimulus, but he indicated vaguely the situation of the contact by rubbing his finger along the assistant's hand, or in four out of eight observations brought it to rest at some more proximal point. Some of these patients say, "The spot seems to be spread out, so that I can't exactly say what part of it you have touched."

Such a condition is in exact harmony with the change in contact sensibility discovered by testing with the graduated hairs. Not only does the touch seem to persist long after the hair has been removed, but it seems as if "something was being dragged across the hand; as if a mosquito was dragging its leg along, not in the least as if you were touching me at one point only." This is the type of disturbance discovered when localization is disordered by a defective reaction of the part to contact stimuli. The touch seems to be spread out, and may be translated into a sensation of movement across the surface.

(c) *Texture.*—The power of recognizing various stuffs by feeling them with the fingers is obviously dependent on the accuracy of tactile impressions. But there is another aspect of this test, which demands the same faculty of appreciating essential similarity and difference underlying the recognition of weight and form. This appeal to two sides of cortical activity can be demonstrated by choosing suitable cases of sensory dissociation. If tactile sensibility is perfect, whilst the tests with various weights and shapes are manifestly affected, the power of recognizing stuffs by their texture will be disturbed in one direction; another sort of defect is found where touch is impaired, and weight and shape can be appreciated without fail.

No. 16 was a characteristic example of the first condition, the more striking, because the whole hand exhibited this form of dissociation. Here, then, was none of that difficulty so often experienced when some fingers are affected, and others are not; the various stuffs could be frankly placed in his hands, and he could handle them freely in the natural manner. The index and little fingers of the two hands responded to a hair of 14 grm./mm.^{2}, and the palms to one of 21 grm./mm.^{2}, showing that tactile sensibility was in no way defective. He was allowed to take each of the following stuffs into his affected hand and to palpate it as he might desire. It was then transferred to the normal hand. Flannel in the left (affected) hand was said to be "something rough," but was recognized at once on the other side. Silk was said to be "smooth, silky," but was called silk in the normal hand; corded velvet and cotton were thought to be "something rough" on the affected side, though recognized as "corded stuff" and "cotton" over normal parts. When a piece of corded velvet was placed in the two hands at the same time, he said: "They are quite different stuff. The piece in the right (normal) hand is corded; it feels rougher and thicker. The piece in the left hand seems smoother and more like calico: I don't think it is silk."

It was not, however, the smoothness and roughness that were not appreciated, but the essential difference which enables us to give each textile material its specific name. It was, in fact, an "agnosia." This view is borne out by the character of his answers to the tests for the recognition of form and of common objects. In every case, with the wooden blocks in the left (affected) hand, he said, "I don't know," though they were perfectly recognized on the normal side. He had no difficulty in naming correctly a knife, penny, sixpence, and scissors in the right palm, but said, "I don't know," when the same objects were placed in the left.

In No. 3, the perfect retention of tactile sensibility made it possible to show how the loss of recognition of weights and textures runs parallel in the three affected fingers.

	Addition and subtraction of weight	Texture
Thumb ..	Perfect. (No mistakes in 12 tests) ..	Perfect recognition.
Index ..	Perfect. (No mistakes in 12 tests) ..	Perfect recognition.
Middle ..	7 right and 5 defective answers ..	2 wrong out of 4. Velvet called "calico," cotton called "flannel."
Ring ..	5 right and 7 defective answers ..	3 "don't know." Flannel named correctly.
Little ..	Gross loss. Inability to tell if there was weight on the hand or not	Gross loss. "Something rough; I can't determine what it is," was his answer to each of the 4 stuffs.

Contrast this progressive agnosia with the series of answers given by No. 13. In this case, there was no difficulty in appreciating the relation between weights, but tactile sensibility was distinctly affected in the same fingers where recognition of texture was disturbed.

	Tactile hairs	Texture
Thumb ..	Perfect record with 21 grm./mm.^{2} R. and L. Not quite perfect in either thumb with 14 grm./mm.^{2}	Perfect answers. No difference between the two hands.
Index ..	Perfect record with 14 grm./mm.^{2} R. and L.	Perfect answers. No difference.
Middle ..	Perfect record on the normal hand with 14 grm./mm.^{2}; imperfect record on affected hand. With 21 grm./mm.^{2} he gave a perfect record R. and L. but said it was "clearer" on the normal side	All answers correct, but he said "It is more difficult on R. (affected), because it is not so sharp, it is duller."
Ring ..	Perfect record on the normal hand with 14 grm./mm.^{2}. Defective answers on the right hand with all hairs of less than 23 grm./mm.^{2}. He said "It doesn't feel like a sharp point at all. It is as if you were touching me with your finger"	None of his answers were quite correct. He said "I can't tell it's a bit of cloth but can't tell if it is rough or smooth in the R. hand so well as in the L."

Patients, in whom the loss of recognition of texture belongs to this class and is associated with want of tactile sensibility, usually attempt to name the stuff placed in their hands; but they frequently add some explanation of the difficulty they recognize in determining the character of the material. They say, for instance, "That is cotton, I think, but I can't feel it so well as in the other hand." "I can always feel the silk, but it is with the rougher materials that I have difficulty."

I think, therefore, that we are justified in concluding that the test for the recognition of various stuffs by their texture depends on two factors in cortical activity: firstly, on the power of appreciating similarity and difference, and, secondly, on an orderly response to contacts of graduated intensity. In the one case, the patient's answer approximates more or less to, "I don't know what it is," whilst in the other he has a greater tendency to hesitate and to be puzzled by the curious sensory differences in the two hands; generally, however, he makes a guess, which would be recorded as a normal answer were it not for the explanation with which it is so frequently qualified.

CHAPTER V.—SUBCORTICAL LESIONS.

We have analysed elsewhere the effect produced by a subcortical lesion on sensation (Head and Holmes [4], p. 174), and such conditions do not materially concern the aims of the present paper. But it is obvious that many gunshot wounds of the brain must injure, more or less severely, other parts than the cortex; some of the signs which appear, even in such carefully selected cases as those I have chosen for my examples of cortical interference, may be due to some disturbance of deeper structures. It is necessary, therefore, to consider shortly those differences in response to the various sensory tests which point to subcortical interference with afferent impulses. Then only will it be possible to determine, in any particular case, the extent to which the disturbance of sensation is due to more superficial or deeper destruction.

The following general rule can be laid down from a study of undoubted subcortical injuries, which have spared both the receptive and essential organs of the optic thalamus. Whenever a stimulus can be appreciated, a threshold can be obtained. For we are dealing with the results produced by interference with incoming impulses, and not with destruction of their receptive centre.

The closer the lesion lies to the internal capsule, the more extensive is usually the loss of sensation, and the more certainly does it tend to assume a hemiplegic distribution. Face, arm and leg are all more or less affected, although one part, such as the hand or foot, may be more gravely disordered than the rest of that half of the body. Thus, although we cannot attribute such cases as I have described in this paper, in every instance, to uncomplicated cortical lesions, we can be certain that they are not due to injury of the afferent fibres of the capsule.

Secondly, the loss of sensation is extremely severe, compared with that caused by more superficial injuries. Stimulation with cotton wool can be appreciated over hair-clad parts, but not over the palm of the hand, or the sole of the foot; for it must be remembered that movement of the hairs makes a potent thalamic appeal. Should either of these parts retain some sensibility to measured tactile stimuli, there is none of that uncertainty seen in cortical cases; the patient does not respond to contact of the lighter test-hairs, and each increase in strength is associated with a progressively larger number of correct answers.

The loss of appreciation of posture and movement is often so severe, that the patient not infrequently fails entirely to recognize the position into which the limb may have been moved passively. When he wakes at night, he has no idea where to find the affected parts, although he is still conscious of the existence of his hand or his foot. Should the disturbance be less severe, the threshold will be raised, but the range of movement required to excite a response can be measured with greater accuracy than is the case with a cortical lesion. Thus, from 15° to 20° may be required before a movement is appreciated, instead of 1° as on the normal side; but all the ten measurements may fall between these limits, instead of ranging widely from 10° to 45°, accompanied by errors of direction and hallucinations, as is so common with injuries of the cortex.

The compass-test usually suffers severely from the coincident disturbance of spacial and tactile impulses. Even when the points are separated to the widest extent possible within one segment of the limb, every application is said to be "just a touch ; they are all the same." Contact is appreciated, but contains no element of "oneness" or "twoness."

Topical localization is gravely defective, as might have been expected from the other aspects assumed by the sensory loss. Here I believe there is a definite tendency to indicate the site of the stimulated spot, not only on wrong digits, but proximally to its true situation. In one case, out of fourteen contacts on various parts of the fingers and thumb, one only was correctly localized; all the others were referred too low upon the digits or to some spot on the dorsum of the hand.

Ice and water at 50° C. can be appreciated without fail in uncomplicated cases of capsular injury, but, not uncommonly, are thought to be "colder" or "hotter" on the affected side. There is, however, no widespread over-reaction so characteristic of lesions that have entirely freed the thalamic centre from cortical control. The loss of thermal sensibility is shown by the want of response to such temperatures as 24° C. and 38° C., and all that lie between them. Upper and lower limits vary in each case, but it is the central portions of the thermal scale that are affected.

The threshold for measured prick is not infrequently somewhat raised; even if it happens to be the same on the two sides, the stimulus is said to be "less sharp" or "blunter" over the affected than over normal parts.

All such faculties as the power of appreciating weight, form, size and texture are lost. The patient can tell when a weight has been added or removed carelessly, so as to produce a jarring contact; but he cannot tell if the remaining pile is lighter or heavier in consequence of the manipulation. He is also conscious that some of the blocks, used in exploring his appreciation of form, have sharp edges and some have none; but he is unable to recognize that they have any shape. Occasionally he knows that some of the tests for texture are rougher, and some are smoother, but he has no idea of their nature.

Here we see the effects of interrupting afferent impulses on their way to the cortex after the point at which they can influence the essential organ of the optic thalamus. In such cases, there is almost always a coincident affection of the pyramidal tract, usually accompanied, at any rate, in the later stages, by some spastic rigidity.

Such a clinical picture differs profoundly from that of the cases on which this paper has been based, both in the character and distribution of the loss of sensation. But it helps us to understand the form assumed by the sensory disturbance when subcortical tissues are affected by the injury, with or without destruction of the grey matter on the surface.

Now it sometimes happens, that a gunshot wound of the skull strikes some part of the brain, outside the primary motor and sensory centres and passes backwards or forwards amongst subcortical structures. Provided the destruction of tissue is limited, and the wound heals well without the formation of a cerebral hernia, it may be possible for the sensory disturbances on the hand to be unequally distributed over the various digits; and yet the character of the reaction to the various tests shows, that the cortical centres have not been destroyed. The character of the loss of sensation is still that which we have learnt to associate with blocking of afferent impulses.

No. 6 was a superb example of this condition. He was wounded on July 3, 1916, in the frontal region well in front of the motor area. I saw him first a fortnight later; but it was in March, 1918, that I had the opportunity of making the extensive series of observations on which are based the following conclusions. At that time, the various digits of his right hand reacted to the measured sensory tests in the following manner.

From this table it is obvious, that the sensory disturbance became steadily worse on passing from the thumb in the direction of the little finger. But it is to the character of the reactions, rather than to the distribution of the defective sensibility, to which I am anxious to call attention. On any one finger, the highest and lowest number of degrees necessary for the appreciation of passive movement lay much

	Little finger	Ring finger	Middle finger	Index finger	Thumb
Measured movement	15° to 20°	10° to 20°	7° to 10°	5° to 10°	2° to 3°
Compass-test	1 cm.	1 cm.	1 cm.	0·5 cm. slower	0·5 cm. "Not so sharp"
Localization	Not affected	Not affected	Not affected	Not affected	Not affected
Tactile hairs	Defective series, even with 110 gr./mm.^{2}	Threshold 110 gr./mm.^{2}	Threshold 70 gr./mm.^{2}	Threshold 35 gr./mm.^{2}	Threshold 35 gr./mm.^{2}
Thermal	Total want of discrimination	3 out of 4 wrong	2 out of 4 wrong	Perfect	Perfect
Prick	9 grm.	7 grm.	7 grm.	6 grm.	3 grm. Normal
Weight	7 out of 12 not appreciated	5 out of 12 not appreciated	Affected	Affected	Normal
Form	Defective	Defective	Hesitation; named correctly	Good	Good
Texture	Complete want of appreciation	3 out of 4 not appreciated	Introspective change	Introspective change	Introspective change

Sensory Condition of the Right Hand in a case of Subcortical Lesion (No. 6).

closer to one another than is the case in pure cortical lesions. It was possible to obtain a true threshold in each digit, unhindered by those inconsequent irregularities and hallucinations of movement which so frequently mar the records in most of the examples illustrating this paper. Throughout the whole series of fifty observations on the various digits, there was but one instance of wrongly named direction; whereas, in cortical cases this is one of the most frequent errors.

Perhaps the most striking difference between the reactions of this patient and those we have noticed in cortical cases was exhibited by the tactile hairs. Not only did the loss of sensibility increase digit by digit with strictly measurable increments, but in every case it was possible to obtain a threshold. Compare such a record as that from the index, ring, or little finger with those obtained in No. 17 (p. 101). To the lighter hairs he made no response; then, as the strength of the stimulus was increased, he began to appreciate the contact, until, with some one of the hairs of sufficient bending strain, a complete series of answers was obtained. We had then passed the maximum threshold. Even on the little finger, where the response was imperfect to the strongest hair that can justifiably be employed, we can trace the steady improvement in the record with each increase of intensity.

	R. (affected)	L (normal)
Index finger—		
14 gr./mm.^{2}	—	iiiiiiiiiiiiiii
14 ,,	No response	
21 ,,	oooooooioooooioo	
23 ,,	ooioiiiiiiiiiiii	
35 ,,	iiiiiiiiiiiiiiii	
Ring finger—		
14 gr./mm.^{2}		iiiiiiiiiiiiiii
14 ,,	No response	
21 ,,	,,	
23 ,,	oooooooooioooooo	
35 ,,	oooooiooiooioioo	
70 ,,	iiiooiiiooiooiii	
110 ,,	iiiiiiiiiiiiiiii	
Little finger—		
14 gr./mm.^{2}		iiiiiiiiiiiiiii
14 ,,	No response	
21 ,,	,,	
23 ,,	,,	
35 ,,	oooiooiooooooooo	
70 ,,	oooooioiooiooioo	
110 ,,	oiiioiioooiooioo	

The records of the tests for appreciation of weight, form, and texture also differed fundamentally from those to which we are accustomed in cases of uncomplicated cortical lesions. When weights were added or subtracted in irregular series, he first complained that it was "not so easy" on the affected finger; then a greater defect was shown by a tendency to miss the removal of a weight, although he did not fail to recognize its addition to the pile. Finally, on the little finger he did not respond to seven out of the twelve additions and subtractions.

The appreciation of texture ran *pari passu* with tactile sensibility, and all the statements of the patient pointed to a strict relation between the two defects.

Recognition of form was, however, perfect in the thumb and index finger; it nowhere showed the gross loss that would have accompanied so considerable a disturbance of the appreciation of weight, had the lesion been cortical.

One of the most striking features was the steadily rising threshold for measured prick, on passing from the thumb towards the little finger. This phenomenon will be dealt with in a subsequent chapter (p. 141).

Another significant aspect of this case, was the affection of the foot. Had the patient suffered from an uncomplicated cortical lesion producing the graded loss of sensibility on the hand, it is unlikely that the lower extremity would have suffered; or it would have formed the central focus of the disturbance to which the condition of the hand was an insignificant accessory. The wide extent of the affection of sensibility, eighteen months after the wound was healed, associated with the comparative slightness of the disturbance at any one part of the field, point to an interference with afferent impulses on their way to the cortex, rather than the destruction of cortical centres.

These records prove that the permanent effect of a high subcortical lesion on sensation is shown by straightforward raising of the threshold rather than by irregular, inconsequent, and uncertain responses. Impulses, which can pass the block, give rise to normal sensations; the patient reacts to the graduated intensity or changing character of the stimulus, whatever its nature.

The closer a subcortical injury lies to the surface of the brain, the more nearly does the character of the sensory loss approximate to that produced by lesions of the grey matter. But the grouping of the disturbed sensibility always tends to diverge more or less from the three categories: recognition of spacial relations, reaction to graduated intensity, and appreciation of the character of the stimulating object. For afferent impulses still remain grouped according to their sensory quality, until they have been subjected to the transforming activities of the cortex.

In each instance of a superficial lesion of the brain, we must analyse the defects of sensation according to these criteria; a definite and orderly progression from complete insensibility to a clear supraliminal response points to subcortical interference with afferent impulses. Irregular answers, inability to come to a conclusion, hallucinations whether of contact or movement, show that the disorder is due mainly to sensory centres. In many cases, the disturbance of sensation shows both these characteristics, a high threshold and irregular response; and there is little doubt that many gunshot wounds of the brain produce their effects by destroying both cortical and subcortical tissues.

CHAPTER VI.—TESTS WHICH APPEAL TO THE CORTEX AND TO THE OPTIC THALAMUS.

§ 1.—*Measured Prick.*

No test is more difficult to devise than a measurable painful stimulus. Pressure is too coarse, and is liable to harm the delicate structures of the skin. Electrical methods are too uncertain and variable in their effect, changing with conditions over which we have no control, such as the conductivity of the superficial tissues. A prick is the only effective means of applying a measurable painful stimulus.

There are, however, several reasons why it is far more untrustworthy and uncertain in its effect than any other measured test amongst those we have habitually employed. I pointed out above, when we were considering the phenomena of punctate sensibility, that the reaction of the pain-spots was extremely variable. The fact that the stimulus, to be effective, must actually puncture the skin makes a long series of observations impossible. After systematic exploration with the point of a sharp needle, the surface structures are injured. This is not the case with heat- and cold-spots, provided suitable temperatures are used in the examination.

A second difficulty arises from the fact that the first effect produced by pricking with a sharp needle is recognition of an acutely pointed object; further pressure adds to this a sensation of pain. It is almost impossible to prevent the patient from saying he is pricked, whenever he appreciates that the stimulus is a sharp point, and, if he is told to wait until he obtains a distinct sensation of pain, we run the risk of placing the threshold too high.

Now this appreciation of the pointed nature of the stimulus is a discriminative faculty which, on the lowest level, is associated with epicritic sensibility. It is bound up with the processes underlying recognition of the size and spacial extent of objects in contact with the surface, and is one of the most potent factors in controlling the diffuse and radiating responses of the protopathic stage of recovery. Thus, from its inception at the periphery, the effect produced by a prick makes a double appeal. On the one hand, the pain it evokes is a function of thalamic activity, whilst the discriminative aspect, the "sense of a point," is the product of cortical centres.

This is evident in cases where the optic thalamus has been freed from cortical control; the threshold for the discomfort produced by pressure is almost always lowered, under favourable conditions, on the affected side. But the threshold for prick is usually somewhat raised, in spite of the great increase in the vividness of the reaction, once it is evoked. The strength of the stimulus may have to be raised from three to eight grammes; when, however, it is increased sufficiently to produce a sensation, the pain and general reaction is enormously greater on the affected side. Evidently something has been lost in acuteness of sensibility by the removal of the activity of the cortex. This is not only that "sense of the point," which has nothing to do even introspectively with pain, but consists of a certain blunting to pain itself. The threshold is raised even to the painful aspect of a prick, whilst it is lowered to the more direct discomfort of excessive pressure.

Evidently, then, the sensory effect produced by a prick owes something to the physiological activity of the cortex. But this is so small, compared with the overwhelming part played by the optic thalamus, that it can only show itself in the records, when a large extent of the cortex is prevented from exercising its sensory functions. This is most easily brought about in practice by destruction of the fibres of the centrum ovale; for in this situation a comparatively small lesion can throw out of play, by undercutting, a large area of the cortex. Thus we find, that subcortical lesions, with or without destruction of the superficial centres, are liable to be associated with a distinct raising of the threshold to prick.

Pure cortical destruction, on the other hand, produces as a rule no true raising of the threshold, unless it is much more extensive than that of the examples cited in this research. It may produce change in the sensation to prick, which the patient recognizes on introspection; the two hands "feel different." He says it is "plainer," "sharper," or "more distinct" over normal parts. Sometimes an intelligent patient will add in explanation, "It is more of a point" on the unaffected side; or "In the good hand it seems like a little wire sticking into me, but in the bad hand it is like the point of a blunt pencil." Sometimes the whole character of the stimulus is said to be changed; "It tingles," "It is like electricity."

Thus we can sum up as follows our present experience of the effect produced by a cerebral injury upon sensations of pricking.

(1) A gross subcortical lesion tends to raise the threshold to measured pricks distinctly, but not to an extreme degree.

(2) Injury to the cortex, uncomplicated by destruction of subcortical tissues, does not tend to raise the threshold materially. But it may produce a profound difference in the character of the sensation; this is due, in great part, to disturbance of that aspect of discrimination sometimes called the "sense of a point."

(3) Cortical activity, however, probably exercises a distinct influence even over the appreciation of the relative intensity of successive pricks. But this is so slight that it was not measurable in most of the cases of injury extending over a comparatively small area which formed the basis of this research.

§ 2.—*Vibration.*

Disease or injury, at lower levels of the nervous system, frequently destroys all power of appreciating the vibration of a tuning-fork. But so grave a loss never occurs in consequence of a stationary lesion of the cortex, unless it happens to be associated with epileptiform attacks or other causes of shock.

This is not to be wondered at, when we consider that a large tuning-fork, beating strongly, is a stimulus to two aspects of sensation. First of all it produces a series of jarring contacts, which appeal to the activity of the optic thalamus; and, as a matter of fact, vibration is appreciated in many cases where the thalamic centre has been freed from cortical control, although the character of the sensation is entirely altered. Secondly, the tuning-fork produces rapidly repeated movements of small range; these can be recognized, provided the appreciation of passive movement is shown to be normal by the usual method of measurement. Thus, in its two aspects, as repeated crude contact stimuli and as a series of small passive movements, vibration makes a double appeal and cannot fail to excite sensation of some kind, unless the block lies in or below the termination of the fillet.

Almost all the cases, on which this research is based, showed some definite shortening of the period during which vibration could be appreciated. Occasionally, the patient volunteered the statement that it was "plainer" or "stronger" over normal than over abnormal parts; but usually he made little comment on any introspective difference between the effect of this test applied to the two hands.

A large tuning-fork, beating 128 vibrations in the second, is brought into contact with the terminal phalanx of one of the fingers, whilst the hand is fully supported on a pillow. The patient is asked to say when the vibration ceases; as soon as he indicates that it is no longer perceived, the fork is transferred to the corresponding portion of the other hand. Under normal conditions the vibration usually becomes appreciable again for a time. The period between the transference of the fork to the other hand and the moment when its beating can no longer be recognized, is measured with a stop-watch. In healthy individuals this may last from five up to fifteen seconds; but a few persons allow the fork to run down so far on its first application that it has ceased to beat before it is transferred. Both these modes of reaction are normal, provided the measured periods are approximately equal from right to left and from left to right. Thus it may happen that, in the one form of normal response, the records read as follows for four observations :—

Right to left	+ 6 sec.		+ 4 sec.	
Left to right		+ 5 sec.		+ 4 sec.

or according to the other mode of reaction,

Right to left	+ 0 sec.		+ 0 sec.	
Left to right		+ 0 sec.		+ 0 sec.

In neither instance was there any material difference between the two hands.

But, whenever sensibility to this test is diminished in consequence of a cortical or subcortical lesion, the period during which vibration is appreciated will be found to be shortened to a greater or less extent on the affected side. Transference from an abnormal part to a similar one on the other half of the body is associated with a prolonged period of renewed appreciation; but, on the contrary, moving the fork from a normal to an affected part leads to no further vibratory sensation.

The records on the next page were obtained in No. 17 by this method.

These figures are typical of the answers obtained in the cases of cortical affection dealt with in this paper. The thumb was obviously normal, but the measurements from the index finger showed a slight shortening, when the fork was transferred from the normal to the abnormal hand. Such small differences lie within the limits of experimental variation; they would have been of no significance had not the readings from the middle finger shown the same tendency in a higher degree. On the ring finger this difference was greatly increased. Finally, over the little finger the defective sensibility to vibration appeared in its extreme form, and it was not appreciated at all when transferred from the normal to the affected hand.

Thumb—				
From R. to L.	+ 8 sec.		+ 9 sec.	
From L. to R.		+ 8 sec.		+ 7 sec.
Index finger—				
From R. to L.	+ 12 sec.		+ 11 sec.	
From L. to R.		+ 9 sec.		+ 8 sec.
Middle finger—				
From R. to L.	+ 10 sec.		+ 10 sec.	
From L. to R.		+ 6 sec.		+ 6 sec.
Ring finger—				
From R. to L.	+ 15 sec.		+ 14 sec.	
From L. to R.		+ 8 sec.		+ 5 sec.
Little finger—				
From R. to L.	+ 15 sec.		+ 10 sec.	
From L. to R.		+ 0 sec.		+ 0 sec.

Evidently the five digits were not in the same condition. The thumb was unaffected, but the disturbance steadily increased in a postaxial direction and reached its maximum over the little finger. When we compare the extent of this loss with that shown by the other means of sensory exploration, it is found to correspond exactly with that revealed by measured movement. To this test also the thumb was normal, the index was somewhat affected, and the middle, ring and little fingers showed grave loss of sensibility; no other method of examination revealed such extensive disturbance.

Throughout all these cases of cortical and subcortical injury, there is the same close correlation between the loss revealed by measured movement and by vibration, not only in the number of digits affected, but also in the gravity of the disturbance. In the one instance of my series, where in spite of other disorders of sensibility passive movements were perfectly appreciated, all the digits of the affected hand gave normal answers to vibration.

In the following table I have combined the records of a series of cases to the two tests. The number of degrees required for recognizing the direction of a passive movement are given in the upper line, whilst the shortened appreciation of vibration on the affected hand is recorded in seconds below. It will be seen how closely the extent of the loss, measured by the number of digits, corresponds with the two tests; at the same time they show a remarkable agreement in the relative degree of the disturbance on any one finger. Individual differences always

Case		Thumb	Index finger	Middle finger	Ring finger	Little finger
No. 10	Movement	1° to 2°	5° to 7°	5° to 10°	Gross	Gross with hallucinations
	Vibration	Shortened	12 sec.	16 sec.	16 sec.	17 sec.
No. 22	Movement	10° to 20°	25° to 40°	35° to 50°	20° to 30°	2° to 3°
	Vibration	14 sec.	16 sec.	18 sec.	12 sec.	No shortening
No. 4	Movement	1° to 3°	3° to 5°. Errors and hallucinations	3° to 5°. Errors and hallucinations	20° to 30°	Absolute loss
	Vibration	6 sec.	About 7 sec.	About 6 sec.	About 7 sec.	Much diminished
No. 20	Movement	3° to 4°	15° to 25°	15° to 25°. Errors	Gross loss	Gross loss
	Vibration	No shortening	5 sec.	9 sec. Irregular	10 sec.	12 sec.
No. 19	Movement	1° to 2°	2° to 3°	4° to 8°	10° to 15°	15° to 20°
	Vibration	2 sec.	5 sec.	8 sec.	8 sec.	8 sec.
No. 3	Movement	1°. Direction bad	2° to 3°	2° to 3°. Slow	Gross loss	Gross loss
	Vibration	7 sec.	8 sec.	10 sec.	10 sec.	10 sec.
No. 7	Movement	1°	2° to 5°	2° to 4°	2° to 4°	10° to 20°. Errors
	Vibration	No shortening	10 sec.	10 sec.	9 sec.	15 sec.
No. 17	Movement	1°	5° to 8°	10° to 20°. Errors	10° to 20°. Errors	7° to 20°. Errors
	Vibration	No shortening	3 sec.	4 sec.	12 sec.	15 sec.
No. 2	Movement	1°	3° to 6°	6° to 10°	5° to 8°	15° to 25°
	Vibration	No shortening	2 sec.	10 sec.	7 sec.	15 sec.
No. 12	Movement	1°	1°	2° to 4°	5° to 10°	15° to 35°. Errors and hallucinations
	Vibration	Doubtful	Injured finger	8 sec.	12 sec.	25 sec.
No. 14	Movement	7° to 10°	4° to 8°	1°	1°	1°
	Vibration	15 sec.	15 sec.	No shortening	No shortening	No shortening

occur, and it must be remembered that the two sets of records are rarely made on the same day. For a set of observations with the tuning-fork must be made when the patient is fresh and free from fatigue.

Both measured movement and vibration, as we have employed it, appeal to the same aspect of cortical sensibility, but for practical purposes the former is the better test; for it is capable of revealing those curious irregularities of response, so characteristic of cortical affections, which appear in the form of hallucinations of movement and positive errors in direction. Moreover, some individuals do not give constant answers to the tuning-fork, as we have applied it, and this tendency may be aggravated in cases of injury to the brain. But, on the other hand, many of the patients with whom I have had to deal in this research responded surprisingly well. They were all young and not infrequently looked upon this test as a game to be played to the full extent of their capacity; in such instances the records showed not only the graduated loss from finger to finger, but remarkable relative constancy on different occasions. But it is all important that the observations should be made alone, in a quiet room, and at the beginning, rather than at the end, of a series of sensory observations.

From the periphery upwards, a normal response to the tuning-fork test has been shown to be associated with impulses arising in the deep afferent system. So long as deep sensibility is intact, vibration can be appreciated; but, during their passage through the spinal cord, all impulses arising in subcutaneous end-organs pass away into secondary tracts, with the exception of those underlying recognition of posture and movement and the vibration of a tuning-fork. A suitable lesion of the posterior columns can abolish all power of responding to these tests.

But, above the point where the fillet ends in the optic thalamus, no lesion produces complete insensibility to vibration, except under conditions of neural shock. We have no right, therefore, to speak of the projection of "deep" sensibility "on the cortex of the brain." Vibration of a tuning-fork is affected by a cortical or subcortical lesion, because it is one method of testing the same faculty which underlies appreciation of passive movements. The power of recognizing a series of minute and rapidly repeated vibratory changes is a function of the discriminative mechanism of the brain.

From the periphery to the cortex, vibration and passive movements evoke sensation or are abolished together. So far I have explained this coincident response to the two tests as a consequence of the basic similarity of the physiological changes on which they depend. For in both of them sensation is evoked by afferent impulses, caused by a series of minute passive movements. But closer consideration shows that there is a deeper cause for their fundamental identity.

Both tests depend on the integrity of the deep afferent system; but any sensory loss due to a peripheral lesion is mixed up with disturbance of other aspects of sensation, which also depend on subcutaneous end-organs. Injury to the posterior columns of the spinal cord may, however, abolish all power of recognizing passive movement and vibration. Tactile sensibility is perfect; the patient can appreciate the lightest touch and frequently is able to recognize that something is happening to that part of the body which is being moved; but the tuning-fork seems to him nothing more than a round flat object pressed upon the skin, and all perception of movement is absent.

Now at first sight it is difficult to see why the repeated jarring contacts of the vibrating fork are not appreciated as a succession of touches, for tactile sensibility is perfect. The manipulations used in testing appreciation of passive movement evoke a series of stresses and varying tactile impressions from joints, tendons and surrounding structures; but in order that these may yield a sensation of movement they must be recognized as an orderly sequence, tending in a certain direction. In the same way, vibration must produce the impression of a series of minute contacts consecutive in time.

Passive movements of any part of the body not only lead to changes in place, but also give the impression of duration in time; we can recognize by introspection that they are taking place in a certain direction and that they occupy a definite period; but the immediate result of such manipulations is the conviction that the limb is being moved in space. On the other hand, vibration is directly translated into its temporal aspect; it produces a series of movements of small range, which give rise primarily to the perception of a sequence in time. It is possible introspectively to recognize that the beating fork produces rapidly repeated contacts; but its dominant psychical effect is recognition of succession in time.

I should like to suggest that both these tests appeal to the same physiological functions. Such a hypothesis is rendered probable by considering the significance of vibration in the life of such a vertebrate as the fish. The organs of the lateral line, so important in the maintenance of posture, respond to vibrations of the water around, and the creature is enabled thereby to maintain its balance in a shifting medium. Here we see the close association between vibration and spacial recognition. But the highest and most perfect of these lateral line organs is the so-called "ear" of a fish. This is the exact homologue of the internal ear of man, but obviously has nothing to do with hearing in the human sense; for it is adapted to pick up and respond to vibrations in the water around. But, like one part of the internal ear in man, it is peculiarly associated with posture. Thus, from a developmental point of view, vibration and changes in posture are closely associated together, and I believe that even in man they evoke the same afferent physiological consequence.

The two forms of movement produce a sensation of orderly sequence both in space and time. But, when the part is moved in one direction passively, the dominant sensation is one of succession in space; when, however, the stimulus is a series of vibrations, we obtain the impression of succession in time. For other sensations tell us that the base of the tuning-fork has remained throughout on the same spot, covering exactly the same area; attention is therefore concentrated on the time aspect of the stimulus rather than on the nature of the minute changes in posture it produces.

The corresponding physiological processes, due to vibration and to passive movement, are translated into a predominant recognition of an orderly series of changes in space or in time, according to the concomitant impulses by which they are attended. From the point of view of somatic sensation, recognition of successive movements in time and in space are the psychical correlatives of the same physiological functions.

CHAPTER VII.—HYPOTONIA.

It is not my intention to attempt a systematic account of the changes in tone produced by lesions of the cortex; but I cannot close this paper without describing that curious hypotonia which so frequently accompanies sensory disturbances of cortical origin. This is the condition to which Guthrie [3] has drawn attention in his paper "On Muscular Atrophy and other Changes in Nutrition associated with Lesions of the Sensory Cortex."

No. 23 was an excellent example, not only of this hypotonia, but of its concomitant phenomena. No photograph can give any but a shadowy indication of the profound difference between his two hands (see p. 237). The affected hand [left] resembled that of a woman, and was soft and rounded with delicate fingers; the right hand was that of a man capable of hard manual work. Its surfaces were squarely modelled, the palm broad and flat, and the fingers had an appearance of shortness in relation to the hand as a whole. The left hand, on the contrary, was rounded and soft; its lines were less deep. The fingers seemed to be longer and thinner than those on the normal side, although by measurement they were actually a little shorter. This illusory appearance was due to the smaller width of the left palm, which measured 8 cm., compared with 9 cm. on the right hand. The tips of the fingers were spatulate on both hands, but less so on the left than on the right, The interosseous spaces were not wasted; they seemed, however, on the affected side to be occupied by smaller and softer muscles. There was no obvious difference in the skin of the two hands, no change in colour and no cyanosis. The patient had done no manual work for four months, and both hands were white and free from corns. The difference between them was immediately evident on contact. In the act of shaking hands, the right grip was firm and well sustained; I was conscious of the elastic pressure of the contracting muscles of the palm and of the tension of the fingers; I had the feeling that, if the patient chose to increase his grasp, it might become painful. But when I gave my left hand into his, it was like the impression received from the hand of a woman. My fingers seemed to be in contact with something soft and without resistance; the pressure was orderly, but gentle and harmless.

All the digits of the left hand could be bent back passively one by one to an abnormal extent; even the thumb seemed to be hypotonic. But the fingers were not paralysed. Individual movements of each digit could be carried out, so long as the eyes were fixed upon them, and the tip of the thumb was approximated without difficulty to that of each finger in turn. But the movements were leisurely and without virility. So long as his eyes were open, the constituent parts of the affected hand remained in alignment with one another, and the thumb did not droop when the limb was extended in front of him.

But, as soon as the eyes were closed, the little finger dropped away from its position, and all the digits tended to fall out of line when abducted. There is nothing specifically diagnostic about this ataxy and defective attitude of the hand; it occurs on closing the eyes whenever the power of recognizing posture is disturbed by a lesion of any suitable part of the central nervous system.

Before passing to consider in closer detail this hypotonia, which so commonly accompanies lesions of the sensory cortex, it is necessary to define the condition with which I intend more particularly to deal. Sensory changes of cortical or subcortical origin are not of necessity associated with hypotonia. In one case, the fingers may be atonic, and in another tone is not diminished, and yet spacial recognition is equally disturbed in both. The key to the difference between the two groups lies in the presence or absence of coincident affection of the motor mechanism. If the disordered sensation is associated with inability to carry out individual movements of the fingers, tone may be normal, or is, at any rate, not diminished apart from gross paralysis. The deep reflexes of the affected limb are usually somewhat increased. Both these defects point to some primary disorder of the motor mechanism additional to, and not consequent on, the disturbance of sensibility.

Now my principal interest in this paper is with those cases where none of these accessory motor or reflex signs were present, and yet hypotonia existed in a distinctive form. For under such conditions the loss of tone is distributed according to the gravity of the sensory loss.

But the hypotonia does not depend indifferently on all aspects of want of sensibility, nor on the incidence of the maximum general disturbance; it is associated peculiarly with defective appreciation of posture and passive movement.

This cannot be proved by such cases as No. 23, for the sensory defects were too widely distributed over the affected hand. But in the case of Lieut. J. [No. 15] the various aspects of sensation were disturbed independently in the two feet, owing to a wound of the vertex. On the right side all spacial tests were well recognized, but tactile appreciation was grossly affected. Here tone was not disturbed. In the left foot, however, the spacial aspects of sensibility were measurably affected, and the great toe was profoundly hypotonic. It could be pushed backwards to such an extent that it came to form less than a right angle with the line of the dorsum of the foot, whilst the right great toe could not be brought up to subtend an angle of less than 120°. In this case, sensation was disturbed on both feet in consequence of a cortical lesion, but the hypotonia was on the side of the loss of spacial appreciation.

Not only is the tonic condition of the hand as a whole dependent on the state of spacial appreciation, but this is true for each individual digit. I have shown that the different parts of the hand may be in a very different sensory condition; some fingers may be grossly affected, whilst others exhibit no change of any kind.

The following table has been constructed to show the close association between the condition of spacial sensibility and the tonic state of the various parts of the hand. The most delicate indication of disturbed appreciation in space is the power of recognizing passive movement. This has been recorded in degrees for each digit in turn and the condition of tone indicated below. It is at once obvious how closely the two physiological states are associated with one another; the fingers, where the loss of spacial sensibility is profound, are at the same time those which are hypotonic.

How closely the amount of hypotonia is associated with the degree of defective spacial appreciation was evident in such a case as No. 12, fifteen months after a bullet wound of the left parietal region. He complained that when he played the piano, there was "a want of purpose" in the ring and little fingers of the right hand; "I cannot rely on them, and they will probably blur the note, unless I watch them very carefully." "These fingers and the corresponding part of the hand," he said, "hardly seem to count, compared with the others, in anything I do."

But he had no difficulty in carrying out isolated movements and approximating the tip of each digit in turn to that of the thumb, provided he was allowed to fix his gaze upon them. When he held out both hands in front of him, even the little and ring fingers were in alignment with the others, so long as his eyes were open; but as soon as they were closed, the two ulnar digits fell away, whilst the thumb and index fingers remained as strictly in position as those of the normal hand.

Now it will be seen from Table A that there was no loss of spacial appreciation in the thumb and index, and a slight defect only in the middle finger; these digits were not hypotonic. But the sensory disturbance was considerable in the ring finger, and in the little it assumed gross proportions; both were hypotonic, but the loss of tone was undoubtedly greater in the latter than in the former, coinciding with the relative degree of loss of sensibility.

This straightforward relation between the loss of postural recognition and diminished static tone can only exist if there is no primary motor defect or disturbance of reflex excitability. Everyone is familiar with the conditions present in a spastic hemiplegia of cortical or subcortical origin. Not only are all the reflexes profoundly exaggerated, but the hand may be incapable even of the coarsest movements. Here the tonic influence of lower centres has been set free to pour uncontrolled

TABLE A.

HYPOTONIA PRESENT.

Case		Thumb	Index finger	Middle finger	Ring finger	Little finger
No. 23	Measured movement ..	10° to 20°	10° to 15°	20° to 30°	15° to 25°	20 to 30°
	Hypotonia	+	+	+	+	+
No. 10	Measured movement ..	1° to 2°	5° to 7°	5° to 10°	Gross loss	Extremely gross loss.
	Hypotonia	Doubtful	+	+	+	++
No 22	Measured movement ..	10° to 20°	25° to 40°	35° to 50°	20° to 30°	2° to 3°
	Hypotonia	+	+	+	+	0
No. 4	Measured movement ..	1° to 3°	3° to 5°	3° to 5°	20° to 30°	Absolute loss.
	Hypotonia	0	+	+	++	+++
No. 19	Measured movement ..	1° to 2°	2° to 3°	4° to 8°	10° to 15°	15° to 20°
	Hypotonia	0	0	+	+	+
No. 5	Measured movement ..	20° to 40°	20° to 40°	10° to 15°	8° to 15°	1°
	Hypotonia	+	+	Doubtful	Doubtful	0
No. 12	Measured movement ..	1°	1°	2° to 4°	5° to 10°	15° to 35° With many errors.
	Hypotonia	0	0	0	+	++
No. 14	Measured movement ..	7° to 10°	4° to 8°	1°	1°	1°
	Hypotonia	?	+	0	0	0
No. 3 (2nd stage)	Measured movement ..	1°	1° to 2°	1° to 3°	Gross loss	Gross loss
	Hypotonia	0	0	0	+	++

into the affected limbs, and at the same time all high-grade voluntary power has been destroyed. Such cases show the combination of paralysis, due to destruction of the motor mechanism of the cortex, combined with tonic overaction of lower centres. It is sometimes possible to overcome this spasticity in the hand by continuous gentle pressure; the rigidity of the fingers yields, and they can then be bent backwards to a degree impossible on the normal hand. Moreover, all the phalanges may become over-extended at the interphalangeal joints, a condition spoken of as "saddle-backed fingers."

But this want of tone, which so often underlies spastic rigidity of high cerebral origin, is part of the paralysis. The whole cortical motor apparatus is disordered; not only is voluntary power destroyed, but the tonic innervation upon which it is based is also diminished. This motor paralysis affects the hand more severely than any other part, because its movements occur habitually under control of the will. Every aspect of voluntary motion is affected; individual movements of the digits are no longer possible, and the static tone which underlies the maintenance of posture is also destroyed. But the clinical picture is complicated by the massive tonic influence of lower centres. Spastic rigidity invades the limb attacking the larger joints to a greater degree than those more habitually under control of the will.

Such conditions do not come directly within the purview of this research; but they form a necessary link in the comprehension of an apparently anomalous group of cases. It sometimes happens that a gunshot injury of the surface of the brain produces loss of recognition of posture and passive movement, without obvious increase or decrease of tone in the fingers. The lesion has not been deep enough, or sufficiently extensive, to produce spastic rigidity. Why then is the loss of spacial recognition not associated with hypotonia?

In all such cases individual movements of the various parts of the hand are clumsy and are performed with difficulty, even when the patient fixes his gaze on the fingers. If he is asked to throw out both hands in front of him the digits are not in perfect alignment, and the thumb may droop abnormally. This is usually more evident if he is made to abduct his fingers and then to bring them together again. Not only is this movement badly performed, but the posture finally assumed is obviously defective. Throughout, the patient has kept his eyes open and directed his gaze on to the affected hand.

Usually, in such cases the deep reflexes do not give an identical response in the two upper extremities; the wrist-jerk is somewhat

TABLE B.

Hypotonia Absent.

Case		Thumb	Index finger	Middle finger	Ring finger	Little finger	Movements and posture	Reflexes
No. 20	Measured movements	3° to 4°	15° to 25°	15° to 25°	Gross loss	Gross loss	Great loss of R. grasp. Individual movements clumsy. Want of alignment	Brisk wrist-jerk R. and L. Other reflexes normal
	Hypotonia	0	0	0	0	0		
No. 11	Measured movements	5° to 7°	6° to 10°	8° to 10°	7° to 10°	7° to 10°	Considerable loss of L. grasp. Individual movements clumsy. Want of alignment	L. wrist- and elbow-jerk greater than R. L. abdominal defective
	Hypotonia	0	0	0	0	0		
No. 3 (1st stage)	Measured movements	1°	2° to 3°	2° to 3°	Gross loss	Gross loss	L. grasp feeble. Individual movements impossible. Want of alignment of little and ring fingers	L. wrist- and elbow-jerk greater than R. L. lower abdominal sluggish
	Hypotonia	0	0	0	0	0		
No. 2	Measured movements	1°	3° to 6°	6° to 10°	5° to 10°	15° to 20° With errors	R. grasp poor. Individual movements defective. Want of alignment of little finger	R. wrist-jerk greater than L. Otherwise reflexes normal
	Hypotonia	0	0	0	0	0		
No. 17	Measured movements	1°	5° to 8°	10° to 20° With errors	10° to 20° With errors	7° to 20° With errors	R. grasp weaker. Individual movements bad. Want of alignment	R. wrist-jerk greater than L. R. lower abdominal defective
	Hypotonia	0	0	0	0	0		

brisker on the affected side and the elbow-jerk may be increased. But we are far from the condition seen in spastic paralysis.

On Table B, I have brought together a set of cases where, at first sight, we might have expected more or less hypotonia of the digits corresponding to the graduated loss of spacial recognition. But, in every instance, individual movements were clumsy and the fingers out of alignment, even though the eyes remained open. The cerebral injury had disordered the motor mechanism of the cortex in addition to its afferent centres.

Defective motor control from the side of the cortex is also shown by the tendency of the wrist-jerk to be brisker on the affected side; but it must be remembered that no deep reflex reveals the state of the various digits. The wrist-jerk indicates the condition of the forearm rather than of the hand, and not uncommonly the difference between these reflexes is very slight in this class of case.

According to the view I have put forward, a lesion of the sensory cortex can produce hypotonia provided the motor activity of the pyramidal system is intact. If, however, the highest grade of voluntary motion is disturbed, the fingers either show no obvious change in tonic innervation, or, if the lesion is more severe, fall into a condition of spastic rigidity or flaccid paralysis.

Should this hypothesis be correct, recovery of motor power without coincident restoration of postural sensibility should lead to the gradual appearance of hypotonia in the affected fingers. This was actually the case in No. 3. He figures on Table B in the first stage in which I saw him; individual movement of the various parts of the left hand could not be carried out, the fingers were out of alignment and the deep reflexes of the upper extremity were exaggerated. At this time his fingers were not hypotonic. But as time went on the movements of the digits improved, and eleven months after the injury he could approximate the tip of the thumb to that of each finger in turn with the eyes open, though more slowly than in the normal hand. Coincidently with this return of movement the little and ring fingers became definitely hypotonic; on the restoration of voluntary power static tone became defective. (See Table A.)

Gross loss of appreciation of posture and passive movement, due to a cerebral lesion, may thus be associated with various states of tonic innervation.

(1) Provided there is no direct interference with the highest motor

functions of the cortex, hypotonia may be directly associated with want of spacial appreciation.

(2) But if, when the eyes remain open, individual movements are clumsy and the fingers are out of alignment, and especially if the deep reflexes point to some interference with the activity of the pyramidal system, no hypotonia may be present.

(3) Should the lesion be sufficiently gross to set free the lower centres from cortical control the limb may be spastic and the fingers rigidly contracted.

(4) Severe lesions of the cortex and subcortical tissues may cause a complete flaccid paralysis, especially in the early stages. Here the fingers are atonic because both voluntary movement and static tone are destroyed by the injury. The phasic and tonic aspects of motion are paralysed.

This paralytic loss of tone may in certain cases exist under cover of general spasticity of the limbs. Once the coarse influx of tone in the hand is overcome by gentle and steady pressure, the fingers can be bent back to an abnormal degree, and the terminal phalanges can be brought into a "saddle-backed" posture.

My business is with the first of these states of tonic innervation; for it alone stands in direct relation to cortical disturbances of sensation. Paralytic atonia and hypertonia deserve a treatise to themselves, and cannot be dealt with within the limits of a paper devoted to the afferent mechanism of the cortex.

All spacial aspects of sensation rise into consciousness influenced by the consequences of some previous experience. For it would be impossible to discover the position of any part of the body, unless the immediate postural sensation was related to something that had preceded it. A direct perception of posture, analogous to that of roughness or of pain, is impossible; every new position of the limb is relative to those which have preceded it.

The sensory cortex is the organ to which we owe the faculty of relating one sensation to another either simultaneously or consecutively. Consequently all spacial aspects of sensation suffer severely from a lesion of the cortex. Not uncommonly a patient with a cortical lesion can recognize that some movement has taken place, but is unable to discover its direction or amplitude; complete appreciation of spacial relationships is the recognition of serial changes in a certain direction.

The cortex has been said to be the repository of "images of movement." This is, however, an unsatisfactory term when we consider the actual effects produced by a cortical lesion; for when we speak of an image we mean something that can be recalled into consciousness under suitable conditions.

When we sit immobile and imagine our fingers touching some object on the table, many of us see at once the mental picture of an outstretched arm; the only image in consciousness is a visual one. Now if we examine an intelligent patient in whom the power of recognizing posture or passive movement is gravely affected, this visual image may remain as vivid as ever. In such cases it is possible to make the following instructive experiment. Place his arm in front of him on the bed, allowing him to see the position in which it lies; touch several spots in succession on the affected hand and ask him to point to them with his finger, his eyes remaining open throughout. Then close his eyes and carefully remove the arm into some distant position, say at right angles to the bed. If he is a strong visualizer he will tell you that he can still see the picture of his arm and hand as vividly as before on the bed in front of him. Now touch some point on the hand; if tactile localization is not too grossly disturbed, the patient will indicate a spot in the position where that part of his hand originally lay on the bed. This phenomenon may be called "exploration of the phantom hand." Here the visual image of the limb remains intact, although the power of appreciating changes in position is abolished.

It is evident therefore that the standard to which immediate reference is made, when a fresh position is recognized, cannot be a visual image. The existence of normal human beings, whose conscious life by day is devoid of all visual images, would be sufficient evidence of this fact, apart even from the direct results of experiment in cases of cortical lesions. Some such persons undoubtedly possess true movement images. That is to say, the assumption of an imagined posture may be accompanied by re-presentation of movement, equivalent to the pictures of those who visualize strongly.

But in both cases the image, whether it be visual or motor, is not the fundamental standard against which all postural changes are measured. Every recognizable change enters into consciousness already charged with its relation to something that has preceded it. Before the afferent processes caused by movement of a joint can evoke a change in consciousness, they have already been integrated and brought into relation with the previous physiological dispositions, due to antecedent postural changes. Just as on a taximeter the measured distance is presented to us already translated into shillings and pence, so the final product of spacial changes rises into consciousness as a measured postural appreciation.

For this standard, against which all subsequent changes of postures are measured before they enter consciousness, we have proposed the word "schema." By means of perpetual alterations in position we are always building up a model of ourselves, which constantly changes. Every new posture or movement is registered on this plastic schema and the activity of the cortex brings each fresh group of sensations evoked by altered posture into relation with it. Immediate postural recognition occurs as soon as this relation is complete.

Recognition of posture and movement is obviously a conscious process. But the activities on which depend the existence and normal character of the schemata lie for ever outside consciousness; they are physiological processes with no direct psychical equivalent. The conduct and habilaments of the actor who appears before us on the stage are the result of activities behind the scenes of which we must remain ignorant, so long as we are only spectators of the play.

The only constant and continuous record of the movements of our bodies in space exists in the condition of our schemata. These physiological dispositions of the cortex ensure that the inception of a voluntary movement will find the part to be moved in a suitable attitude. For, unless postural impulses perpetually modified these unconscious physiological activities consonantly with every change in position, we might will a certain movement although the limb was not in a suitable attitude to bring it into being. This is evident in every case of ataxy.

But in order that the part of the body to be moved may be ready to spring off, like a runner, at the word of command, its static tone must be normal. Should it be posed atonically, the voluntary motor act has first to gather in slack before the limb begins to move. Normal posture and normal tone are coincident terms.

It is obvious therefore that any lesion which disturbs postural schemata will interfere with static tone. For, in order that a part of the body at rest may retain a normal posture, afferent postural impulses must exert a constant influence on the activity of the appropriate receptive centres of the cortex. These are the repository of spacial schemata. The physiological changes brought about by this stream of afferent impressions not only checks and controls voluntary movement, but ensures that the static tone of the part shall be adapted to maintain its position. Consciousness is in no way necessary for such co-ordination; in fact the regulation of tonic innervation occurs entirely on the physiological level. Any lesion, which tends to destroy postural schemata, not only disorders voluntary movement, but under suitable conditions may diminish static tone.

In order that this disturbing effect may show itself in a permanent diminution of tone when the part is at rest, the pyramidal system must be intact. For, if the motor mechanism of the cortex is directly affected, fine regulation of postural tone is no longer possible; the general tonic condition of the part tends to be increased, as shown by exaggeration of its deep reflexes. The more profound the lesion the grosser becomes this spastic rigidity due to the uncontrolled massive innervation from lower centres.

But the hypotonia, so closely associated with defective recognition of posture and passive movement in the absence of any defect in the highest motor apparatus, is in reality a false co-ordination. The receptive centres of the cortex are badly informed of the position of the affected parts; at the same time those physiological dispositions past and present, on which depend the existence of spacial schemata, are disordered. Voluntary movement is badly co-ordinated except when guided by vision. But tonic innervation is a purely automatic process, not under the control of the will except in so far as it is the aftermath of a voluntary movement. Static tone consequently suffers and the affected fingers are more or less permanently hypotonic.

I have laid stress throughout on this twofold influence of a cortical lesion on spacial recognition. A subcortical injury may also disturb recognition of posture by cutting off, to a considerable extent, afferent impulses from the receptive centres in the cortex. In such a case the threshold is raised, but it may be possible to obtain orderly and coherent responses if the intensity of the stimulus is sufficiently increased. Thus it may be necessary to move the finger passively through an angle of 20° before appreciation occurs, but all the answers will tend to lie round about this degree. If, however, the cortex itself is destroyed it may be impossible to obtain a true movement threshold. Not only is the range at which appreciation occurs widely different, but false answers and hallucinations mar the records. We are now dealing not only with the disturbance of afferent impulses, but with the destruction of the centre for those physiological processes which underlie the recognition of posture and movement.

This difference between the consequences of a pure subcortical lesion and one which involves the cortex is, I believe, also manifest in the condition of static tone. The former leaves the permanent dispositions of the afferent centres intact; the patient still possesses complete spacial schemata, but incoming influences are defective. The internal organization is intact, but it is badly served with information from without. Now, static tone is automatic; it is the product of a constant stream of influence exerted by the simple existence of the physiological dispositions we have called schemata. So long as they remain intact, tone will not be grossly diminished, apart from the effects of a primary paralysis. Once, however, the inherent activities of the afferent centres are destroyed by a cortical lesion, tone tends to be diminished because of the loss of the constant automatic influence they exert on the highest efferent mechanism.

This is one of the reasons why a pure subcortical lesion, such as that in No. 6 (p. 137), was not associated with hypotonia of the fingers. We might at first sight have anticipated a graduated diminution of tone increasing towards the post-axial aspect of the hand corresponding to the want of recognition of passive movement. But all the signs pointed to an uncomplicated lesion of the centrum ovale as far as the post-central cortex was concerned. A threshold could be obtained to all the sensory tests, although to most of them it was raised considerably above the normal. The answers were free from the gross irregularities and inconstancy so common when the sensory cortex is itself affected. Here the normal condition of static tone was due to the continued exercise of that co-ordinating influence, which constantly flows from the highest afferent to the efferent centres.

Thus, it is evident that the so-called sensory cortex exercises by its very existence a double effect upon motor activity. First of all it controls voluntary movement; conscious appreciation of relations of the body in space go hand in hand with the effector activities of the will. But these phenomena of mind are based on physiological processes which may be directly disturbed by a cortical lesion. It is these underlying non-mental dispositions which influence postural tone. Thus, at this anatomical situation in the nervous system, the vital activity of the centre is expressed as presentations to consciousness, concerned with the position of the body in space, and also as the purely physiological condition of postural tone.

CHAPTER VIII.—THE PRINCIPLES WHICH DETERMINE ANATOMICAL LOCALIZATION OF SENSORY FUNCTIONS IN THE CORTEX.

No one now denies that sensation can be affected by a lesion of the cerebral cortex, and the disputes, which raged over this question, can be relegated to the past history of medicine. Moreover, it is certain that the "excito-motor" area, that portion which gives origin to the pyramidal tract, is situated almost entirely within the precentral convolution; it is not coterminous with the so-called "sensory cortex." For, although the anterior and posterior limits of the sensory centres are still undetermined, everyone is now agreed that the post-central convolutions form the most effective focus, from which sensory changes can be evoked by a suitable injury to the brain.

But preoccupation with the practical problems of topographical localization blinded neurologists to the true nature of the functions, which are disturbed by a cortical lesion. They assumed that all disorders of sensation would fall into the categories of touch, pain, heat and cold, together with some "spacial" or "muscle-sense" constituent, and were surprised to find that researches on these lines ended in confusion.

Russel and Horsley ([14] p. 139) frankly stated in 1906 that "all observers are agreed that isolated lesions of the Rolandic zone do not produce complete tactile anæsthesia or analgesia (to these must be added temperature anæsthesia)"; but they offered no solution of this apparently anomalous conclusion, and did not state definitely in what way sensibility was disturbed by a lesion of the cortex.

Recently, however, some authorities on cortical topography have attempted to avoid this difficulty by adopting a most disastrous nomenclature. Thus v. Monakow speaks of disturbances of "deep, proprioceptive, protopathic and epicritic sensibility" in connexion with cortical lesions ([12] p. 270-271). Even so recently as 1916 van Valkenburg [17] published a paper entitled, "Sensibilitätsspaltung nach dem Hinterstrangtypus in Folge von Herden der Regio rolandica."

Dejerine [2] in 1914 wrote as follows: "Une autre question se pose depuis quelque temps; on a constaté dans plusieurs cas d'hémanesthésie cérébrale une topographie spéciale de l'anesthésie, qui se traduit sous forme de bandes reproduisant plus ou moins exactement la distribution radiculaire spinale. En présence de ces faits, on est amené à se demander s'il n'existe pas dans les centres corticaux sensitifs une systématisation radiculaire comparable à celle qui existe pour les centres moteurs

corticaux." Here, it is true, he did not state that the nature of the loss of sensibility corresponded to that caused by injury of the posterior roots; he only asserted that the distribution of the disturbance might follow a radicular type. But, as a matter of fact, the anatomical form and the essential character of any loss of sensibility are closely bound together; when it follows a peripheral, radicular, or spinal distribution, the nature of the sensory change corresponds to the situation at which it is produced. To suppose that a cortical lesion could be associated with a sensory loss of radicular form is as absurd as to postulate a spinal lesion with an ulnar nerve paralysis.

I have attempted to show that sensory changes, produced by a lesion of the cortex, must be considered in terms of the psychical act and not of the physical stimulus. The function of the cortex in sensation is to endow it with spacial relationships, with the power of responding in a graduated manner to stimuli of different intensities, and with those qualities by which we recognize the similarity or difference of objects brought into contact with the body. It is these aspects of sensibility which are disturbed by cortical injuries. The anatomical lesion upsets the orderly sequence of physiological processes and this defect of function is manifested in disorder of the psychical act of sensation. The terms in which this disturbance is manifested are categories of of the function itself; a sensory loss of cortical origin is not expressed in anatomical or physiological terms, but in those of the most elementary processes of mind.

If the view I have put forward in this paper is correct all previous theories of cortical localization, as far as sensation is concerned, must be put aside. The two conceptions are mutually antagonistic and cannot be combined; there is no *via media*. Unless this fundamental diversity of outlook is recognized from the start, it will be impossible for the reader to grasp the attitude I have assumed with regard to the topographical significance of sensory disorders of cortical origin.

We may accept as a sufficiently well observed fact, that a lesion of the post-central cortex can produce a profound effect on sensibility. But we have no right to assume that those forms of sensation, which are disturbed, are of necessity functions of the injured cortex. For any sudden injury of the surface of the brain produces widely radiating shock effects within the structures of the nervous system. Sometimes a blow on the head, or a cerebral operation, may be followed by loss of sensation so severe that it is difficult to believe the mid-brain has not been affected. The whole of one half of the body may become insensitive to touch, pain, heat and cold; and yet the rapidity with which this loss of sensation disappears shows that it was not due to gross structural changes in the lower afferent centres.

An epileptiform attack is a potent cause of neural shock, and in the attempt to determine the sensory function of the cortex, it is better, if possible, to reject those patients who suffer from any form of seizure or periodic disturbance of consciousness. No demonstrative value can be laid on sensory changes which follow directly on a Jacksonian convulsion.

This widespread consequence of a cerebral injury or convulsive attack has been called "diaschisis" by von Monakow ([11] p. 237). He believes that the shock is so great that it passes backwards, over one or more synaptic junctions, to affect the functions of anatomical systems remote from the site of the injury. He adds: "Es handelt sich auch bei der Diaschisis im wesentlichen um Herabsetzung oder Aufhebung der Anspruchsfähigkeit . . . der zentralen Elemente für Reize innerhalb eines bestimmten physiologisch wohl definierten Erregungskreises; dieser Kreis fällt indessen mit dem gewöhnlichen, von der Peripherie und vom Zentrum aus sich ausdehnenden *physiologischen* Innervationswege *nicht* zusammen."

Now, whatever we may think of this exclusively anatomical standpoint, the widespread effect of a strictly local injury of the cortex is undoubted. It is impossible to say, in the early days of a wound of the head, how much of the loss of sensation is due to destruction at the site of the lesion and how much to irradiation. The profoundness of the effect is even more disconcerting than its wide extent. Not only is sensation disturbed over a larger area of the body than might have been expected from the situation of the injury, but the character of the loss differs fundamentally from that found with a stationary lesion of the cortex. Slowly, day by day, sensation returns to those parts of the body which do not correspond to the focus of disturbance. At the same time, the nature of the sensory loss changes; it no longer consists of a gross defect of tactile, painful and thermal sensibility, but, in favourable cases, begins to assume the characters I have described in this paper.

All aspects of cerebral activity are liable to undergo similar profound and extensive changes in consequence of trauma; these are particularly evident in the phenomena which follow cerebral concussion. At first the patient lies in flaccid unconsciousness, with shallow breathing, scarcely able to swallow fluid placed in his mouth. Then the lower centres recover from the shock and he becomes restless and even violent. As normal consciousness returns he grows amenable to control and his conduct is no longer dominated by the activities of the lower functional levels.

In the same way, on the sensory side, the gross effects produced by the initial lesion, or by a subsequent operation, gradually pass away. If the wound heals well and there are no epileptiform attacks, all the initial loss of sensation may disappear. But, if the lesion lies over the post-central convolution, some defect of sensibility may remain for months or years in that part of the body which corresponds to the focus of the lesion. This loss is never completely hemiplegic, provided the cortex alone is injured; it occupies a part only of the opposite half of the body and may even be confined to a few digits on the hand.

It is most instructive to watch the order in which the sensory loss gradually diminishes. The outlying parts become normal and sensibility is defective in those parts only, which formed the focus of the previous disturbance. Thus in No. 23 the whole hand was affected; in addition the records at the wrist, elbow and great toe originally showed some loss of recognition of passive movement. These three joints became normal within six months of the injury. During this recovery the sole of the foot regained its sensibility to the tactile hairs, which had been profoundly disturbed. Coincidently with the gradual restriction of the affected area came a diminution in the severity of the sensory loss on the various parts of the hand; the amount of the loss to measured movement and to the tactile hairs became less, even over those fingers which were most affected.

But when once the wound has firmly healed, particularly if the opening in the skull is small and does not pulsate, it is remarkable how constant may be the extent and character of the sensory changes. Such are the cases which form ideal material for determining the part played by the cortex in sensation. Much can, however, be learnt from less perfect examples, provided care is taken to eliminate the effect of shock and other transitory consequences of cerebral injury.

§ 1.—*Representation of Parts of the Body.*

Many of the tests I have used are not applicable to any part but the hand; for instance, the power of discriminating different weights and textile fabrics can be explored over the feet in the rarest instances only.

Even such a universal test as the compass-points does not yield equivalent results on the hand and on the foot. For, on the palmar aspect of the tips of the fingers, the two points applied at the same moment can be recognized in many cases, when they are separated by 0·5 cm.; whilst on the sole of the foot it is not possible to depend on correct answers at a smaller distance than about 2·5 to 3 cm. Thus, if the power of discrimination were reduced to one half, good readings might be obtained on the hand at 1 cm., but on the foot we should be compelled to separate the two points to a distance of at least 5 to 6 cm. In the same way the range of this test is greatly limited on the forearm, where it is impossible in a normal person to be certain of obtaining a complete series of correct answers under about 4 cm. Over such parts the two points must be separated so widely, before we can obtain even a normal threshold, that the limits within which the test can be applied are rapidly exceeded, whenever sensibility is affected to any considerable degree. Again, it must not be forgotten that the sole of the foot is the equivalent of the palm of the hand and not of the fingers. As we cannot use the compass-test with any satisfactory results on the toes, we are unable to explore the condition of the distal segments of the lower extremity; the first evidence of changed sensibility must be drawn from the penultimate parts of the limb.

Similar difficulties apply to all the other tests, and the results obtained from the feet are always less distinctive and coarser than those from the hand. As far as the face is concerned, most of them are quite inapplicable. It is impossible to test the power of appreciating passive movement, or recognition of weight, form and the situation of the stimulated spot. Fortunately, however, in the voluntary movements of the face and tongue, we possess a delicate measure of any loss of motor power that may be caused by the lesion. By this means it is often possible to decide whether the main impact of the injury has fallen on the centre for the face; if so, the sensory changes in the hand may be due to its radiating effect and if so should ultimately disappear slowly. If they are still present at the end of six months, they are probably caused by some permanent destruction of cortical tissue affecting the arm-centre, although the main incidence of the lesion has fallen on that for the face.

In the same way, the sensory changes in the hand may be accessory to a more severe disturbance of function in the foot. Passive movements may be badly recognized in the great toe, at the ankle and even at the knee; the compass-points may not be appreciated at 10 cm. on the sole, and no threshold obtained with the tactile hairs. Obviously, in such a case the sensory changes are extremely gross and the cortical centre for the foot has been badly injured. But the loss of sensation in the hand may occupy one or two digits only and be of a more delicate character; the various forms of sensory activity may be dissociated, one may be lost over the affected parts, whilst another is entirely unaffected. In such a case it is evident that the hand does not lie in the central focus of the disturbance.

Thus, for all delicate exploration of the sensory faculties of the body, we are thrown back on to the hand. Sometimes, in the cases I have used for this research, the disturbance of sensation is the result of direct injury to that part of the sensory cortex in which the hand is topographically represented; in other instances it is due to the outlying effect of a lesion, that has fallen either on to the centre for the leg or on to that for the face. This makes any precise determination of the limits of these centres extremely difficult, from such material as I have brought forward in this paper.

But there can be little doubt that the higher the lesion on the central convolutions the more certainly will the lower extremity be affected, whilst the closer it lies to the Sylvian fissure the more definite will be the affection of the face. Thus when an injury affects both the face and hand we should expect it to lie lower on the surface of the hemisphere than when it disturbed the functions of the hand and foot. This is actually the case.

It is now generally recognized that a lesion of the post-central convolution is more likely to lead to definite and severe disturbances of sensation than of any other part of the cortex. Here those physiological activities, which underlie sensation, are concentrated more thickly than at any other part of surface of the brain. The sensory condition of the hand or the foot is more or less gravely disturbed, whenever the incidence of the injury falls upon this area. If the middle portion of the convolution is struck, and the destruction of tissue is not severe, the hand alone may be affected; but the nearer the lesion lies to the vertex, the more certainly will the foot participate in the disturbance. If it happens to lie on the vertical aspect of the hemisphere, any affection of the hand that may be present will almost certainly be less severe than the loss of function in the lower extremity. Conversely, when the injury falls upon the lower half of the post-central convolution, some affection of the face and tongue is likely to accompany the loss of sensation in the hand. Evidently the sensory activities of the hand are more closely represented in the middle of the convolution.

But it is possible to determine more in detail the topographical representation of the hand upon the cortex. This research has been based upon cases where the various fingers were not all in the same sensory condition; sometimes the pre-axial, sometimes the post-axial digits were most severely affected. Now whenever the loss of sensation reaches its maximum over the thumb and index finger, the face and tongue tend to be affected; if the lesion lies in the left hemisphere, speech will also have been disturbed, to a greater or less extent, at some time in the history of the patient. This is shown by the five cases in Table C where the impact of the wound lay mainly over the central convolutions. If, however, the loss of sensation was greatest over the little finger and its neighbouring digits, it is the foot which tends to show some motor or sensory change. Out of seven such cases, four showed some disturbance of the lower extremity, although in most instances it was slight and transient; but movements of the face and tongue were normal in all and speech had not been affected. (Table D.) I do not intend for a moment to assert that, because the little finger shows the profoundest changes in sensibility, the face and tongue cannot suffer; one of the commonest results of a wound of the skull in the region of the central convolutions is to produce a more or less transitory hemiplegia. But it is not with these cases that I am dealing at present; my interest is centred in those where the various digits were in a widely different sensory condition. These are the less severe cases. It is among them that we tend to find this association between the pre-axial parts of the hand and some affection of the face and tongue; or, conversely, a tendency for the functions of the foot to be disturbed, when the main loss of sensation lay over the little finger.

Moreover, even the rough verbal description of the locality of the injury, given in the last column of the table, brings out the same point. For in those cases where the maximum incidence of the loss of sensation lay over the little finger, the lesion was usually situated higher on the post-central convolution than when the thumb and index were most affected. Thus it is evident that the pre-axial digits are more definitely represented in the lower part of the centre for the hand, whilst the post-axial portions lie topographically higher on the surface of the hemisphere.

But it must not be supposed that the maximum loss of sensation fell either over the thumb or over the little finger in every instance. Four of my cases showed preponderating affection of the little finger;

in six the disturbance reached its greatest intensity over the little and ring fingers; in one it was more severe over the thumb and in three others over the thumb and index finger; in one case the most intense sensory loss was found in the index and in another it was greatest in the index and middle fingers. Finally, in one instance, it reached its maximum over the middle finger.

TABLE C.

Case	Thumb	Index finger	Middle finger	Ring finger	Little finger	Affection of face	Affection of foot	Locality of the lesion
No. 9	+	++	++	0	0	Speech and face at first	0	Middle pre-central
No. 13	+	++	Introspective	Introspective Very slight	Introspective	Speech at first; face weak; tongue to R.	0	Inferior pre-central and post-central
No. 14	++	+	0	0	0	Speech and face	0	Middle pre- and post-central
No. 18	++	++	Slight	0	0	Face affected; tongue to L.	0	Inferior pre- and post-central
No. 11	++	+++	+	Introspective	Introspective	Speech at first; face and tongue slightly	0	Middle post-central

TABLE D.

Case	Thumb	Index finger	Middle finger	Ring finger	Little finger	Affection of face	Affection of foot	Locality of the lesion
No. 2	0	Slight	+	+	+++	0	0	Middle post-central
No. 3	0	? very slight	Slight	++	++	0	Leg affected at first	,, ,,
No. 4	Introspective	+	++	+++	+++	0	Leg slightly affected	,, ,,
No. 7	+	+	+	+	+++	0	Definitely affected	Upper pre-central and post-central
No. 12	0	0	+	+	+++	0	0	Upper post-central and upper parietal lobule
No. 19	0	Slight	+	++	++	0	0	Middle pre-central and post-central
No. 20	Slight	++	++	+++	+++	0	Slight weakness and sensory change	Middle post-central

Thus, it is evident that the various fingers are represented topographically in the cortical centres; sometimes one and sometimes another lies in the focus of the disturbance. It is convenient to speak of the thumb in this connexion as pre-axial and of the little finger as post-axial; but it must be clearly understood that the axis of the limb is not represented topographically on the cortex. Certain motor and sensory functions of the body are more concentrated at some parts of the hemisphere than at others. In the same way, the physiological activities corresponding to each separate digit are relatively more highly represented in a certain portion of the cortical centre for the hand. But axial lines are a conception drawn from developmental anatomy; they have no place in the functions of a limb free to move in space. From the point of view of sensation, anatomical representation ceases as soon as the first synaptic junction is passed. The body is not represented on the cortex as an anatomical unity, but only in so far as it is capable of undergoing conscious changes in function. Thus, all attempts to establish a local representation of such purely anatomical factors as axial lines are based upon a false conception of the nature of cortical activity, and of the significance of central representation.

Russel and Horsley [14] were not content with speaking of topographical localization of the pre- and post-axial halves of the limb, but even postulated the "representation in the cerebral pallium of the mid-axis of the upper limb" (p. 149): This conception they based on no more valid evidence than sensory changes "in the two middle fingers of the hand." This, they say, seemed to them "such an obvious corollary to the pre-axial and post-axial representation we have just discussed, that we bring forward the following case as a good example of the clinical fact." This case showed nothing more, however, than an "aura beginning in the third and fourth fingers of the left hand, spreading to arm, face, trunk and leg of ths same side," and seventy-three days after removal of a large subcortical tumour, anæsthesia to cotton wool on the dorsum of the hand with tactile localization "towards the mid-axis."

No better example could be given of the difference between the older methods of thought and the conceptions I have attempted to put forward in this paper. Sensation is a psychical act, and even the regional representation of the processes on which it depends is based on functional habits, rather than on phylogenetic development. Each digit is a unit and can be used in an isolated act, either of motion or sensation; as such it is represented topographically on the cortex.

The same rule applies to loss of sensation on the palm of the hand; the distribution of the changes in sensibility bears no relation to axial lines, but corresponds to the proximal extension of the affected digits. Many of the tests I have applied to the fingers cannot be employed on the palm; but the compasses, tactile hairs and weights can all be used over limited areas. If, then, the palm is marked out by lines running from the four fingers in the direction of the fold of the wrist, whilst the greater part of the thenar eminence is reserved for the thumb, it is found that the sensory condition of the intervening portions closely resembles that of the digits to which they correspond.

This is particularly evident in such cases as No. 3, where the compass-test was disturbed over the post-axial digits, but tactile sensibility was entirely unaffected. This dissociation both in the extent and quality of the sensory loss was equally manifest on the palm. Over the thenar eminence, and in a line with the index finger, his answers to the compasses applied simultaneously were perfect; but over the post-axial portion of the palm they were grossly disturbed, even when the two points were separated by a distance of 5 cm. At the same time, tactile sensibility was everywhere normal.

In No. 9 the index and middle fingers showed the most severe loss of sensation, but the ring and little fingers were unaffected. On the pre-axial side of a line, drawn vertically down the palm from the cleft between the third and fourth digits, he was unable to appreciate weights correctly, just as on the corresponding fingers. But, on the ulnar half of the palm, sensation was entirely unaffected to this test. In other cases the tactile hairs revealed exactly analogous changes.

Thus, it would appear as if the loss of sensibility on the palm, produced by a cortical lesion, corresponds roughly both in situation and quality with that of the affected digits. There are no "axial" lines or borders; as the sensory condition changes from finger to finger, so it alters both in quality and intensity on passing from one part of the palm to another.

When sensation is disturbed over the whole of the hand, it may be defective at the wrist, elbow, or even at the shoulder. Under these circumstances the foot usually participates in the sensory loss. This is particularly liable to occur when we have reason to believe, from other signs and symptoms, that subcortical tissues have been affected; in fact, the deeper the lesion the more certainly will the sensory loss assume a hemiplegic distribution.

Occasionally, however, both hand and foot may be normal to measured tests, and yet some more proximal part of the upper or lower extremity may show definite loss of sensibility. But these cases are rare. No. 8 is a particularly good example, because not only have we definite evidence that both hand and foot were little, if at all, affected at an early stage in the progress of the case, but I saw him for the first time six weeks after he was wounded and could discover no gross changes, except at the elbow and to a less degree at the wrist. Recognition of passive movement was profoundly disturbed and the tactile hairs also revealed considerable sensory loss over the forearm. No measurable defect could be discovered in the palm and digits; introspectively, however, he found it more difficult to make up his mind with certainty over the hand of the affected than of the sound limb. The lower extremity was in every way normal. In this case the elbow evidently lay in the focus of the cortical disturbance, and the wrist suffered to a less degree.

All experimental evidence points to the topographical representation of the various movements of any one limb in different parts of the cortex (Sherrington and Grünbaum [16]). Of all the centres associated with the upper extremity, that for the fingers and thumb lies lowest on the precentral convolution; then follow in order those for the wrist, elbow and shoulder. Between the shoulder and the hip, lies the area devoted to movements of the trunk.

Now, I have already shown that, from a sensory point of view, a lesion which affects the thumb and little finger is liable to be accompanied by some defect in speech or disordered movement of the face and tongue. Thus of all the centres devoted to the physiological activities underlying sensation in the hand, that for the thumb must lie lowest on the surface of the brain. Conversely, the foot occasionally shows some loss of sensibility, when the maximum disturbance lies over the little finger; this digit must therefore be represented in the highest part of the cortical centre for the hand.

It follows, as a corollary to this conclusion, that, whenever the little finger shows gross changes in sensibility, the wrist also tends to be affected. Should the whole hand be involved, but the maximum loss lie over its post-axial parts, not only the wrist, but the elbow and shoulder, may show some defective recognition of posture and passive movements.

So far I have shown that the disturbance of sensation produced by a cortical or subcortical lesion tends to follow a regional distribution corresponding to the contiguity of the centres on the cortex. But it is the functions, rather than the anatomical relationships, of any one part of the body that are represented. Consequently those portions, such as the hand, which are endowed with the greatest powers of discriminative sensibility, are represented, and to the highest degree, in the sensory cortex; for the fingers and thumb of man are little more than stalked sense-organs. Next in order comes the sole of the foot, which constantly exerts discriminative activity in walking. One of my patients (No. 6), in whom the right foot was affected, said: "When I am in the dark, I always take my feeling from my left foot, whereas before I always used to test the ground with my right. I have changed over since I was wounded."

In consequence of this more exclusive representation, the hand and the foot may suffer, although the sensory functions of the intervening joints are not measurably disturbed. In No. 20, for example, all the digits were affected, but the grossest loss of sensibility was found in the little and ring fingers; the wrist showed slight want of recognition of passive movements, but the elbow and shoulder were normal. In spite of the escape of these two joints, the great toe was affected and the sole of the foot showed an introspective difference to the tactile hairs. In a similar way it is possible for the great toe and the sole to show sensory changes, and for the little finger to be affected, although passive movements are perfectly appreciated at the ankle.

Thus, although the cortical centres are arranged according to the relative position of the different parts of the body, a lesion of a definite area does not, of necessity, disturb sensation in this anatomical order; hand and foot may suffer without obvious loss at the elbow and shoulder. The extent of cortical representation depends on the degree to which function is developed. The hand, with its five mobile digits, stands at the head of this functional hierarchy. It is therefore most highly and most widely represented in the sensory cortex. It is important to remember that even the regional representation of parts of the body, as revealed by a cortical lesion, is governed by the degree of functional development rather than by purely anatomical considerations.

§ 2.—*Representation of Sensory Functions.*

The part played by the cortex in sensation is concerned with those physiological processes which underlie projection and discrimination. I have attempted to show that these activities fall into three groups; they are represented on the sensory side by appreciation of relationships in space, by appropriate reactions to stimuli of different intensities, and by recognition of the similarity and difference in test-objects of various weights and shapes.

We saw reason to believe that these three aspects of cortical activity were not always affected uniformly, or in any constant relation to one another. In one case the spacial tests yielded normal results, in spite of considerable defects in the remaining aspects of sensation; whilst in some instances the tactile hairs, or in others the graduated weights, failed to show any abnormality.

These are, however, extreme examples, but they demonstrate that the three aspects of sensibility cannot be equally represented in all parts of the sensory cortex. Even cases where the dissociation is less complete point in the same direction; for it is not uncommon to find that one group of tests shows a disturbance of sensibility in five digits, whilst another group reveals defects in one finger only.

These three faculties might have stood to one another in some such relation of graduated complexity as that which we discovered for appreciation of tests in three-, two-, and one-dimensional space. The cortex is the organ for controlling and guiding voluntary movement, and recognition of the relations of the body in space is one of its most important functions. It would not, therefore, have been unreasonable to suppose that whatever the changes on the afferent side produced by a lesion of the cortex, this aspect of sensibility would suffer. To a certain extent this is true. But at the same time the existence of one such case as No. 7, where the spacial tests gave normal results, shows that the physiological processes underlying the three forms of sensibility can be completely dissociated by injury to different parts of the cortex. They must then be based on processes which can be disturbed independently.

Moreover, if the three functions stood in a graduated relation to one another, one aspect of sensation would always show a more severe and more extensive disturbance than either of the others. But this is not the case; sometimes one group of tests, sometimes another, reveals a disturbance of greatest severity and widest extent. If we take passive movement, the tactile hairs and graduated weights as representative means for exploring the three sides of cortical sensibility. Table E shows to what extent they may be affected independently of one another.

Such extreme dissociation shows that the maximum activity of all three physiological processes cannot be situated within the same region of the sensory cortex. Each one must be represented more highly at some part than at another; in this way it may sometimes be more severely affected than its fellows, and at others may escape entirely. But any attempt to demonstrate the locality of the centres for the various aspects of sensibility is hampered by the extreme difficulty of determining the incidence of the lesion. One wound extends deeply and produces widely radiating changes, both anatomical and physiological; another, less severe, in an identical situation, may be followed, after the first shock has passed away, by changes in sensation which reveal the functions at the site of the lesion. Not only is it difficult to gauge the extent and severity of the original injury, but we can rarely tell how far the activity of the surgeon extended amongst the structures of the brain itself. All cases are not of equal value, from the point of view of topographical localization. Some showed evidence of severe destruction of tissue; they had been trephined, the dura was found to have been ruptured and disintegrated brain was removed at the operation. In others the skull had not been opened and may not even have been fractured; and yet there were obvious signs that the cortex had been structurally affected.

TABLE E.

Case	Measured movement	Tactile hairs	Weights	Situation of lesions
No. 18	0	3 digits	2 digits	Inferior post- and pre-central touching Sylvian fissure. (Trephined, cerebral substance removed)
No. 3	5 digits	0	2 digits	Middle post-central. (Trephined, extradural blood-clot removed)
No. 21	Whole hand and up to elbow	0	Defective	Middle pre-central. (Trephined; no notes, but on arrival dura open and brain substance protruding)
No. 16	Whole hand and upper extremity	0	Gross loss over whole hand	Middle post-central. (Very small removal of bone. Dura lacerated; hæmorrhage from vessel of dura)
No. 2	4 digits	0 (1 digit introspective)	1 digit (slow and introspective)	Middle post-central. (Not trephined)
No. 19	5 digits	2 digits slightly (1 digit introspective)	0	Middle pre-central, post-central and superior parietal lobule. (Trephined; brain pulp removed)
No. 13	4 digits (3 slight only)	5	0	Inferior pre- and post-central, very low. (Trephined; blood-clot and ruptured brain matter removed)
No. 22	5 digits	3 introspective only	0	Pre-central extending into middle post-central. (Small trephined area. No notes)
No. 14	2 digits	2 digits	0	Middle pre- and post-central. (Not trephined)

All attempts to localize the site of the various forms of sensory activity, with the help of such material, must be purely tentative. But by comparing the cases where one function was gravely affected with those where it had escaped entirely, we arrive at certain general conclusions.

First of all, we are justified in asserting that a lesion situated anywhere within a certain area on the hemisphere will disturb somatic sensibility to a greater or less extent; this is the so-called "sensory cortex." It consists of the pre- and post-central convolutions, the anterior part of the superior parietal lobule and the angular gyri. Loss of sensation may show itself in the hand even though the lesion lies low on the pre- or post-central convolutions in a position associated with the centre of the face. Conversely, when it is situated so high as to come within the area usually assigned to the lower extremity, the hand may show characteristic sensory abnormalities. But, whenever the main incidence of the lesion falls on those parts which contain the centres for either the leg or the face, the character of the sensory loss in the hand tends to be peculiar; one form or another of sensation may be entirely unaffected or show comparatively slight alteration.

Injury anywhere within this "sensory area" on the hemisphere is liable to cause some loss of appreciation of spacial relationship in the hand or in the foot. But the further forward it lies within this region, the grosser and more extensive is the disturbance shown by the spacial tests. Thus in No. 21, where the whole hand and elbow showed want of recognition of passive movements, the lesion lay over the middle of the pre-central convolution; but tactile sensibility was completely unaffected. If the middle of the post-central convolution is struck by the injury the spacial aspects of sensibility are usually profoundly diminished in the hand; but some other faculty is also disturbed at the same time.

The power of recognizing differences in weight and form is most severely and most extensively affected by a lesion, which falls mainly over the post-central convolution. But these tests can rarely be applied to the foot with any satisfactory result; for this reason we are only justified in saying that, if the lesion lies over the middle of the post-central convolution, the faculty of appreciating similarity and difference in objects of various weights and shapes, placed in the hand, will be more or less profoundly affected (No. 3, No. 16, No. 20). Among the patients in whom this form of discrimination was not disturbed the lesion was mainly pre-central in two (No. 13, No. 22); in two other cases it lay in the supra-marginal region and the loss was extremely slight (No. 10, No. 23). In all four instances the injury fell either mainly in front of or behind the post-central convolution.

The most surprising conclusion is reached in those cases where the power of responding appropriately to stimuli of graduated intensity was impaired. The most frequent consequences of a lesion within the "sensory area" of the cortex is some loss of recognition of spacial relationships and of similarity and difference; this is the well-known group of symptoms sometimes called "astereognosis and atopognosis." It is notorious that in many cortical cases tactile sensibility is not disturbed, even when we employ such fine tests as the graduated hairs. But in other instances this faculty is profoundly affected; in No. 10 the loss exceeded that of any other form of sensibility, and here the lesion lay over the supra-marginal convolution. The power of responding adequately to stimuli of graduated intensity was diminished, throughout my series of cases, whenever the focus of the injury lay over the superior parietal lobule (No. 5), over the supra-marginal region (No. 10), or over the foot of the post- and pre-central convolutions (No. 13 and No. 18). It is noticeable in other instances that, when the lesion was situated over the lower part of the central convolutions, tactile sensibility tended to show characteristic changes, even though other forms might also be profoundly disturbed. But the higher it lies on the hemisphere and the further forward its situation, the less will be the affection of tactile sensibility. Thus it is possible to construct a zone, running backwards along the foot of the pre- and post-central convolutions, over the angular and supra-marginal gyri to the superior parietal lobule. Any lesion which falls mainly within this area will probably be associated with disturbance of tactile sensibility; if this is severe and extensive, the patient may also be unable to distinguish various degrees of warmth about which he has no doubt on the normal hand.

Thus, the three aspects of sensibility, which are due to cortical activity, are not all equally represented in different parts of the sensory cortex. The spacial tests are most profoundly affected, when the lesion affects the pre-central convolution. The faculty of appreciating similarity and difference, in objects of different shapes and weights, suffers most with an injury of the post-central convolution; whilst the power of responding to the various degrees of intensity of such stimuli as the graduated hairs or warm test-tubes is most certainly diminished by a lesion of the parts behind the post-central convolution and also its extreme foot. That is to say, the centre for the spacial elements lies mainly in front, that for response to varying intensities behind, and that for recognition of similarity and difference between them. At the same time these roughly vertical areas fall somewhat obliquely, so that the representation of the thumb is much farther forwards than that of the foot.

CHAPTER IX.—GENERAL CONCLUSIONS.[1]

§ 1.—*The Significance of Dissociated Sensibility.*

We have been able to show, that between the impact of a physical stimulus and the sensation it evokes, a multitude of changes occur, which do not normally enter consciousness. These are essentially physiological and can only be discovered by some form of dissociation.

The day of the *a priori* psychologist is over as far as sensation is concerned. A man can no longer sit in his study and spin out of himself the laws of psychology by a process of self-examination. For we have been able to show that, at a level deeper than any he can reach by introspection, are prepared those states, which condition the nature and characteristics of the ultimate sensation. Appreciation of position in space, graduated response to stimuli of varying intensity, and recognition of the similarity and difference of objects in contact with the body, were all thought to be matters of "judgment." We have shown that the forms assumed by these aspects of sensation are ordered and predestined on the physiological level, as the result of innumerable integrations, which take place outside consciousness. These processes are not open to conscious analysis; it is only the interplay of sensations that can be discovered by introspection.

The psychologist, who attempts to discover a strict psychophysical parallelism, ignores the central link of the problem. He assumes that the nature and conditions of the physical stimulus can be brought into direct relation with the psychical act of sensation. Light can be analysed by the spectroscope into certain constituents, and he attempts to establish a strict correlation between these "primary colours" and elementary visual sensations. This fundamental error vitiates much of the work on the psychology of the senses. It is only under artificial conditions, that the physiologist can foretell exactly what reaction will follow a given physical stimulus; previous occurrences in the tissue may entirely change the nature of the response. Adaptation, biphasic activity and facilitation form a normal part of the vital activity of the nervous system; they may intervene between a measured physical stimulus and the physiological effect, and make it impossible to establish a direct and immediate correlation.

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[1] It will be evident that most of the ideas expressed in this chapter are based upon the theories of Hughlings Jackson (especially [10]) and the brilliant experiments of Sherrington [15], but I have thought it better not to interrupt my exposition in order to attribute to their originators conceptions, that form the basis of our present neurological knowledge.

Therefore, between the impact of some physical force on the tissues of the body and the psychical act of sensation, are interposed reactions, which, in many cases, cannot be predicted. What wonder that those, who examined the phenomena of the senses at first hand, were dissatisfied with current psychological teaching. The neurologists were driven back more and more on to an examination of changes in structure. They found that, when certain conducting paths were destroyed, sensation was disordered in a definite manner. From this they justly concluded, that the affected paths carried certain specific impulses; and in this they were strictly within their rights. Out of this examination of the loss of sensibility, caused by lesions of the spinal cord and brain-stem, grew the dissociation method, which is the basis of this research.

But many workers on these lines failed to recognize the significance of the method they employed. They discovered that dissociation occurred as a consequence of injury to various tracts in the spinal cord; for instance, sensibility to pain, to heat or to cold may be disturbed as an isolated manifestation of a lesion of the lateral columns. This is due to interference with the conduction of impulses, which have already undergone a certain amount of qualitative integration. It is the most easily determined form of afferent dissociation, and the results seemed to coincide closely with the conceptions usually held of the natural divisions of sensation.

Lesions of the spinal cord can abolish sensibility to pain, heat, and cold, and any one of these qualities of sensation may be lost, although the others remain more or less undisturbed. But this is not the case with lesions, either of the peripheral nervous system, or of the cortex; they produce changes which cannot be classified strictly in this manner. There is a tendency, therefore, to confine the term "dissociation" to those cases, where sensibility is lost to touch, to pain, to heat or to cold, either in an isolated form or in some specific combination. Other sensory changes are spoken of as a "hypo-æsthesia"; and the French include all modifications of sensibility, except anæsthesia and hyper-æsthesia, in the term "paræsthesia" ([2] p. 785).

But under both these expressions lies the false assumption, that afferent impulses are isolated specific reactions to four categories of sensory experience. Let us then avoid the use of these question-begging terms, and in each case determine the exact form assumed by the abnormal response. The researches summarized in this paper show, that the sensory elements revealed by dissociation differ according to the site of the lesion. Division of the peripheral nerves, at any rate in the limbs, reveals the specific characters of deep and superficial sensibility. Whatever may be the phylogenetic significance of this dissociation, it corresponds in man mainly to a difference in the situation of end-organs and in the course of afferent fibres. In the spinal cord this peripheral grouping has been broken up ; dissociation reveals, on the one hand qualitatively distinct groups of impulses travelling in secondary paths, on the other it may expose the primary spacial and discriminative elements of sensibility, which travel up uncrossed in the posterior columns of the spinal cord.

At each anatomical situation in the central nervous system, dissociation assumes forms appropriate to the functional combinations of afferent impulses at that point. Finally a lesion of the cortex causes changes which can be expressed in psychical terms only. The physiological substrata of sensation are disturbed, and the defects appear as want of discrimination. This is not a "hypo-æsthesia" or "paræsthesia," but a definite loss of power to recognize spacial relationships, graduated intensity, and similarity or difference in objects in contact with the body.

Analysis of afferent impulses, on their passage through the central nervous system, tells us nothing of their origin or final fate. If we wish to learn how they arose, we must produce an analogous dissociation in the peripheral nervous system; and their ultimate grouping can only be shown by breaking up those complex physiological activities, which directly underlie sensation. At either end of the physiological chain, we are brought face to face with elementary processes. At the periphery, we see how a measurable physical stimulus is transformed into various physiological reactions. At the other end we can observe the ultimate condition of these impulses, in the act of subserving sensation, after they have undergone integration and selection.

Now the grouping of afferent impulses, on their way from the periphery to the highest physiological centres, is of considerable theoretical interest and profound diagnostic importance. But it reveals little or nothing of the reactions of the sensitive tissues to the impact of physical forces, or of the physiological basis of sensation. Consequently, whilst the practical neurologist tends to concentrate his attention on the mode of conduction, the physiologist and psychologist are interested primarily in the conditions at the two ends of the physiological chain.

It is therefore important that we should appreciate clearly the significance of the facts of dissociated sensibility. We have laid down that the superficial structures of the body are innervated by two mechanisms, one of which we say exhibits more primitive characters than the other. Removal of epicritic sensibility exposes the activity of the protopathic system in its full nakedness. In the same way, when removal of the influence of the cortex cerebri sets free the optic thalamus from control, sensation assumes an overwhelmingly "thalamic" character.

In each case a lower, more primitive organization, is permanently kept under control by the activity of a higher afferent system. But removal of this dominant mechanism does not reveal the functions of the phylogenetically older organs in all their primary simplicity. The original thalamencephalon contained elements, not only of the human optic thalamus, but also, in a crude form, of certain physiological processes, now entirely relegated to the cerebral cortex. Even the specific activity of this original thalamus was incomparably less highly developed than the dissociated thalamic functions of man. A lesion, which sets free the human optic thalamus, produces a highly specialized series of phenomena, which have never existed in this form in phylogenetic history. For a sensory reaction is the product of the activity of two great receptive centres; as we ascend the biological scale, each of these two physiological organs takes over more exclusively the initiation of certain aspects of sensation.

The setting free of a primitive activity from higher control reveals a condition, which is a part of the complete act and does not reproduce an ancient mechanism in its original form. This is evident in such a reaction as the "mass-reflex," which may make its appearance, under favourable conditions, when the human spinal cord has been completely divided [5]. On pricking almost any part below the lesion, the movements evoked consist of flexion of the lower extremity at all joints, accompanied by an upward movement of the great toe. No primary extension occurs under such conditions, but the reaction is always a defensive withdrawal of the part from stimulation. But this is not the only effect; the bladder and rectum are excited to evacuate their contents prematurely, and a profuse outburst of sweating may be evoked by pricking the sole of the foot. Local signature has been abolished, and the response has assumed a massive and primitive character. Normally, this old protective reaction is dominated by centres in the mid-brain; its flexor movements are employed as an element in the assumption of posture, and the overflow of energy into the visceral system is inhibited from above. When this control is cut off, by division of the spinal cord, flexion occurs alone; but it now appears in its primitive form, as a massive reflex response, accompanied by visceral activity. This reaction is more perfectly and specifically flexor, than that which occurs in the dog under similar circumstances. For, in the lower animals, the specialization of function is less complete, and, when dissociation occurs, the separated spinal cord still retains certain functions, which in man have been assumed by higher centres. No flexor response is so free from postural characters, as that obtained from the human spinal centres, after complete division of the cord. In the same way, the reactions of the optic thalamus in man, freed from cortical control, are an almost perfect expression of the non-discriminative aspects of sensation.

In this way many lower activities are retained in a controlled form and are not abolished in the course of evolution, because they may be required, at some time or another, for their primitive purpose. Under abnormal conditions, painful impulses of visceral origin can break through the normal resistance against them and appear in consciousness as a sensation. If so, the distribution of the areas of tenderness represents an old segmental arrangement of the central nervous system, and the reflexes, especially of the abdomen, take on a protective character. This is also the case with the curious diffuse sensation, which alone represents superficial tactile sensibility over protopathic parts. It is normally dominated by more discriminative aspects of touch, but is not abolished; for, under suitable conditions, even in normal life, it may appear as a diffuse and intolerable itching.

Pain is the oldest defensive reaction, and potentially painful stimuli are the basis of all primitive reflexes. It is, therefore, of importance for higher development, that these impulses should be rendered less effective in favour of those impressions which can lead to a more discriminative response. But, although they are controlled and even abolished, the mechanism underlying the production of pain must remain in full physiological activity, ready to play its part, should occasion arise, in the defence of the body against noxious influences.

The functions of the central nervous system are not a palimpsest where a new text is written over an earlier manuscript partly erased. The more primitive activities have been profoundly modified by the advent of the new centres, which utilize some of the faculties originally possessed by the older mechanism. In many cases the higher function could not be exercised without the existence of those lower powers which it dominates and controls. Postural reflexes, strictly adapted to conditions of space and time, could not occur without the outburst of energy which occurs in lower spinal centres on peripheral stimulation. The afferent impulses, which pour into the cerebellar system from the limbs, can only manifest their existence through the lower motor activities they co-ordinate. In the same way, the part played by the sensory cortex is based upon the activities of the optic thalamus; without crude sensations of heat and cold, discrimination would be impossible, and the finer thermal relations could not be appreciated.

When dissociation occurs, either on the reflex or psychical level, the manifestations represent that portion of the complete function which still remains active. If the factor, which has been eliminated, exercised some control over that which remains, primitive characters may appear which are not manifest under normal conditions. Should the function lost belong to the same level as that which remains, the loss of the one does not materially affect the other. But, when one of them is higher in the functional hierarchy than the other, the removal of the dominant influence sets free the lower activities to manifest themselves unchecked.

§ 2.—*Integration.*

Between the impact of a physical stimulus on the peripheral end-organs of the nervous system, and the simplest changes it evokes in consciousness, lie the various phases of physiological activity. The diverse effects produced on the living organism by a stimulus are sorted, combined and inhibited in their passage through the central nervous system and the ultimate results finally act upon their appropriate centres. This process of integration takes place apart from any direct participation of consciousness; and, by the time afferent impulses reach one of those centres, where they can form the underlying basis of sensation, they have been profoundly modified. Such integration is the essential task of the central nervous system; it is carried out by the exercise of certain functional activities, which I shall now consider from their theoretical aspect.

We are accustomed to think of the external stimuli we employ in scientific investigation, as if they were simple physical conditions; thus we speak of the effects produced by "a temperature of 45° C." But in reality the stimulus is always an external object, which evokes a multitude of physiological reactions; it is not only warm, but has certain tactile values, and a definite size, shape and texture. From the physiological point of view, a simple stimulus is an abstraction and an external object is a group of functional events.

Each aspect of the complex stimuli of daily life acts on one or more peripheral end-organs; in this way, the physiological response is rendered more or less specific at its inception. From the first impact of a hot or cold object on the skin, the presence of the heat- and cold-spots ensures that under normal conditions, the reaction will be specific. Any temperature, which excites the cold-spots, generates a set of afferent impulses capable of evoking a sensation of cold. Whatever their ultimate fate, they are stamped from the beginning with a certain character, for they are capable of exciting the activity of those terminal physiological centres which subserve thermal sensibility.

So far, the conception we have put forward corresponds to the original doctrine of specific nerve energy. The older investigators believed that an impulse, once started in specific end-organs, travelled unchanged to the highest receptive centres, to evoke a response corresponding in quality and degree to the sensory attributes of the physical stimulus. Such a theory ignores the fact that a temperature of 45° C. is an effective stimulus to the heat-, the cold-, and the pain-spots, and yet, under normal conditions, produces a sensation of pleasant warmth only. In such a case, a definite physical condition excites three separate groups of end-organs. The impulses evoked are specific in quality; excitation of the cold-spots produces a sensation of cold, although the stimulus is a temperature of 45° C., and even 40° C. can evoke pain in the absence of the dominant thermal mechanism.

The simplest physical condition acting on the peripheral nervous system, may produce afferent impulses which are incompatible from the point of view of sensation. But the stimuli of daily life are not simple; they are extremely complex, and give rise to a multitude of diverse impressions. These must be sorted and regrouped; some are facilitated, others are repressed before the final sum is presented to consciousness. Many afferent impulses remain on the physiological level and never form the basis of a sensation; they are destined to control reflex activity, or to co-ordinate movements of the body and limbs.

Such integration is the task of the central nervous system, and it is carried out by means of a series of receptors, which guard certain synaptic junctions on the centripetal paths. These are in reality end-organs, exposed to the influence of the complex mass of afferent impulses generated in the peripheral nervous system by the impact of an external stimulus. They are not influenced directly by the forces of the world around; they react to the diverse physiological conditions produced by the action of physical stimuli on the receptive mechanism at the periphery. Like resonators placed in a concert hall, each group picks up those qualities to which it is attuned, and refuses those with which it is not in harmony. This process is repeated throughout the central nervous system, until the final products of integration come to act on two terminal physiological centres, the cortex and the optic thalamus. Here they excite those conditions which underlie the more discriminative or more affective aspects of sensation.

But we must not forget that the larger number of afferent impulses, arising in peripheral end-organs, do not traverse the whole length of the central nervous system. Their essential task is to excite and co-ordinate reflex action without of necessity giving rise to sensation, and the earlier phases of integration are undoubtedly adapted to this end. Sensation, in the strict sense of the term, demands the existence of consciousness and is, therefore, a late development in phylogenetic history. On the other hand, reflex adaptation is one of the earliest manifestations of multicellular existence, and the central nervous system is pre-eminently an organization for controlling reflex activities.

Such co-ordination could not occur, if there was no qualitative selection of afferent impulses at the receptive junctions of the various reflex arcs. The main function of intramedullary receptors is to lower the excitability of the reflex arc for one kind of stimulation, and to heighten it for all others. This ensures a selective response to the complex results of peripheral excitation, and at the same time precludes many afferent impulses from influencing consciousness. Thus, for example, visceral insensibility is a function of the specific reaction of receptors, and not the consequence of an absence of afferent impulses.

Each specific group of end-organs, within the central nervous system, works on the same principles; it accepts those elements of a massive reaction which can excite it to activity, and rejects those to which it cannot respond. At the same time there is evidence to show, that the synaptic junctions are subject to those refractory states and phases of heightened receptivity characteristic of all neural action.

These three processes, selection of certain impulses, rejection of others, together with biphasic states of the receptive mechanism, are sufficient to produce coherent reactions on reflex levels. The movements evoked by a stimulus bear a strict relation to its quality and intensity; they are also influenced by the conditions to which the reflex arc has been previously exposed. Thus, pricking and pressure, applied to the sole of the foot, produce different responses; and the nature of the antecedent reflexes is of fundamental importance in determining the form of the reaction to stimuli of the same quality and strength.

At this level of neural activity, all functions are simple, but at the same time highly organized. The response is rigidly pre-determined within certain limits; there is little choice, and the answer is physiologically inevitable. In strict accordance with such an arrangement, we find that the first sorting of afferent impulses within the spinal cord is concerned mainly with their redistribution according to quality. Those capable of exciting pain are gathered together, whatever their peripheral origin, and all those which can evoke sensations of heat are combined into a specific group.

After these multitudinous impulses have carried out their reflex and co-ordinating functions, the remainder are destined to act upon those centres which underlie sensation. Here the rigidity of response, so characteristic of reflex activity, would be disastrous. The limited freedom of the lower levels must be expanded, so that the physiological reaction may become less inevitable and more elastic; for we are dealing with impulses which are capable, under favourable conditions, of exciting consciousness. But the sensations to which they would naturally give rise are incompatible, and cannot exist simultaneously. Thus 45° C. is capable of producing sensations of heat, of cold, or of pain. If they could all coexist in consciousness, mental activity would be chaotic and discrimination impossible. The struggle between these various impulses takes place, therefore, on the physiological level, and the victor alone appears in consciousness as a sensation.

The simplest sensory act is the result, not only of prolonged qualitative integration, but of a constant struggle for physiological dominance. In the example I have just cited the answer is under normal conditions inevitable. But there are many cases, especially within the visceral field, where no general rule can be laid down; the response depends on the patient's idiosyncrasy or general resistance. This may be naturally low to certain afferent impressions and high to others. Moreover, his usual reaction may become changed in consequence of some functional alteration in the activity of the central nervous system; impulses may then excite sensation, which would normally have remained outside consciousness in its widest sense.

On the lowest levels of reflex activity, an impulse that is inhibited and rendered non-effective is to a great extent abolished. It may perhaps exert some influence in determining the future behaviour of the receptive synapse; but it is not manifested in any direct and immediate change. But on the higher physiological planes, impulses, which are precluded from exciting sensation, are not wiped out; they may produce a profound and manifest effect, although they cannot excite consciousness. The postural impulses, ascending in the posterior columns, normally affect two terminal centres. They reach the cortex and evoke sensations of the position of various parts of the body in space, and at the same time pass to the cerebellum to aid in the co-ordination of voluntary movement. But if they are prevented from reaching the cortex, the patient may be entirely ignorant of the position of his affected limbs, and yet the motions of these parts are perfectly co-ordinated. The sensory aspect of these impulses has been abolished, but their unconscious activity remains unimpaired.

The closer we come to those centres, which directly underlie sensation, the more evident is it that impulses, which fail to excite consciousness, do not of necessity cease to exert any physiological influence. They may be repressed by their successful rivals, but they are not therefore abolished. At any moment they may manifest their existence if the dominant influence is removed. In the absence of the afferent heat mechanism on the periphery, 45° C. produces a sensation of cold; if in addition the skin is devoid of cold-spots, the same stimulus evokes pain. Under normal conditions, there is nothing to prevent this temperature from seeming cold, except the prepotent activity of the impulses which are capable of exciting a sensation of heat. In the same way this stimulus produces impulses that can cause pain; but these are dominated by that feeling of pleasure usually associated with warmth of this degree. If, however, for some reason, no coincident thermal impulses reach the physiological centres, or if the patient's resistance to painful impressions is low, 45° C. may cause pain with its customary disagreeable feeling-tone. There is no inherent structural block on the passage of these painful impulses. Temperatures as low as 40° C. undoubtedly stimulate the pain-spots; but the impulses so produced fail to excite consciousness, because they are repressed by the other sensory effects evoked by the same external stimulus. This is even more evident on the psychical plane, when we are dealing with the interplay of two or more highly organized sensations, or other fully developed mental processes.

Sherrington has firmly established the influence of previous occurrences in a reflex arc on the nature of its immediate response. This action of the past, in the event of the moment, is even more evident when the afferent impulse excites, not a reflex but a sensation. The phenomena of adaptation owe their existence to this factor. Thus, under favourable conditions, a temperature of 30° C. not uncommonly seems neutral to the normal hand. If, however, it has been soaked in hot water for some time, a tube at 30° C. may appear to be cold. Conversely, if the hand has been adapted to cold water, the same temperature, that seemed at one time neutral and at another cold, is now thought to be warm. The external stimulus is identical, but the reaction is fundamentally changed, owing to the diverse dispositions created by occurrences in the past.

The effective influence of the past on the reaction of the moment is even more evident in the recognition of spacial relationships. These sensations enter consciousness already measured against a postural standard, built up out of previous movements, and subject to constant change. The dispositions on which this standard depends exist on the physiological level, and a normal sensation of posture is based on activities that are integrated outside consciousness.

From the lowest co-ordinated reflexes to the psychical processes of sensation, discrimination, and feeling, the part played by the activity of the central nervous system is the same, It ensures that impulses of similar quality facilitate one another. Conversely, if two incompatible impulses are excited together, one is allowed to pass, whilst the other is rejected during one of the many integrations, either at a low or high level of functional activity. Thirdly, the vital processes of the nervous system are responsible for the phenomena of adaptation. No stimulus, however mechanically unvaried, can evoke a constant response, because the very existence of the impulses it produces changes the disposition of the reacting centre.

The effect of these three simple processes is evident at every level of neural function, from the lowest to the highest. If we trace a group of afferent impulses from their origin to the final sensations they excite, we find at each stage that the response is given in terms of the function exercised at each particular level. Thus the effect produced by a set of postural afferents is first manifested in the spacial co-ordination of reflexes. Higher in the nervous system their influence is seen in the control of posture and regulation of tone. Finally they evoke recognition of the position and movement of the body in three-dimensional space. But, whether the product of their activity be physiological or psychical, the same three fundamental processes are evident.

If, then, a receptive centre is thrown out of activity by some mechanical injury, the loss of function will be expressed negatively in terms of that level. But the positive manifestations will represent the various influences that can still be exerted by the blocked impulses. Those that can no longer express themselves psychically as a sensation can still exert their action on co-ordination and the reflexes.

The same physiological processes underlie the afferent integration of a reflex and the co-ordination of two sensations or even of two concepts. But, in each instance, the product of these vital functions is given in the terms of the level at which the impulses exert their influence; in the one case it is expressed reflexly, in the other it is manifested in some psychical act, such as sensation.

§ 3.—*Projected Sensations.*

In the less highly developed forms of animal life the body is composed of many segments, each of which retains a considerable degree of autonomy. With the process of evolution the headward parts become increasingly dominant; they contain what Sherrington calls the "distance receptors", organs, such as the eye, associated peculiarly with projected sensations.

As far as somatic sensibility is concerned, it is the body and limbs which contain the "distance receptors"; but the cerebral cortex forms the highest physiological centre for all but the simplest projectional aspects of sensation. When the influence of the cortex is removed, and the optic thalamus exerts its activity uncontrolled, the patient may cease to associate his sensory experiences with any external agency. He complains that he is being hurt, or that something is happening to him, but fails to recognize that he is being pricked with a pin. Removal of the cortical factors in sensation has reduced to elementary proportions the power of projection, as we know it in the intact human being. It is no longer possible to recognize the size, shape, weight, and spacial relations of an external object, nor, indeed, to appreciate the relative intensity of the stimulating action it excites.

All these aspects of sensation are said to be projected; we attribute them to something inherent in the external object. The affective consequences of a stimulus betray its relation to ourselves; it is pleasant or unpleasant, salutary or harmful. But the projected sensations reveal to us the world without. They are responsible for recognition of our relations in space; to them we owe conscious appreciation of degrees of sensory intensity and the power to appraise the similarity or difference of external objects.

These factors in sensation do not depend primarily on "judgment" or "association"; for, on the physiological level, afferent impulses possess projectional characteristics. The same class of impressions which underlie discriminative sensibility can be discovered at work controlling and regulating purely unconscious actions. Analysis of the reflexes, even in man, shows that the character of the response depends not only on the quality, but on the position of the stimulated spot; scratching the sole of the foot is followed by a different movement from that caused by stimulating the skin of the thigh.

Study of the reflexes at higher functional levels shows that they are governed by remarkably complex projectional relations. So purposeful and varied is the response that it is difficult to believe the movement is not controlled by the will; and yet the patient may be ignorant that anything has happened to him. The movement is not only adjusted to the locality of the excitation and the position of the limb, but depends on the intensity and character of the stimulus. The nature of the response is determined, not so much by the absolute intensity and quality of the stimulus as by the relation it bears to those which have preceded it in time, or are in action simultaneously; the capacity of a stimulus to evoke a particular reaction depends on its relations in the past or in the present. Thus, on the reflex level, afferent impulses can be shown to be adapted to spacial conditions and to the intensity and relative character of the stimulus, although the whole procedure remains outside consciousness. If, however, they succeed in reaching the highest receptive centres, they endow sensation with spacial attributes, relative intensity, and individual character.

Evidently, therefore, the impressions produced on a sentient surface by an external stimulus are endowed with certain sensory potentialities from their origin as physiological reactions. The immediate consequence of the impact of some physical force is the production of an incoherent mass of afferent impressions, which must undergo integration before they can exercise any useful function. This is true not only for their qualitative, but also for their projectional characters.

The simplest projected aspect of sensation is shown in the power to localize the site of the stimulated spot on the surface of the body. But observations on my hand showed that the faculty was the product of a struggle between various different impulses with diverse sensory potentialities. During the earlier stages of the experiment, when the surface was entirely insensitive, all sensations were due to excitation of the end-organs of the deep afferent system which had not been affected by the lesion. At this time any tactile stimulus, applied with sufficient force to excite a sensation, was localized with remarkable accuracy. If the back of my hand was pressed with some hard object, such as a pencil, I was able to indicate the spot without difficulty. But with the return of protopathic sensibility to the surface, a struggle arose between the different forms of projection characteristic of the two different systems. Any stimulus capable of evoking a cutaneous response aroused a widespread sensation, referred to some remote part of the affected area. Brushing the back of the hand with cotton wool, in the neighbourhood of the index knuckle, produced a tingling over the whole dorsal aspect of the thumb. Not only was the sensation localized in a part distant from the point of excitation, but it seemed to be spread over an extensive area; this bore little direct relation to the size of the stimulated surface. This erroneous localization was not fortuitous, but remained the same whatever the stimulus employed, provided it was effective; heat, cold, and pricking, applied in the neighbourhood of my index knuckle, all evoked diffuse sensations on the back of the thumb.

If, however, the stimulating object was capable of arousing a sensory reaction in both the protopathic and deep afferent systems, a struggle occurred between the two forms of projection. Suppose, for example, that a tube containing ice-water was applied to the affected area; excitation of the cold-spots produced a massive, diffuse sensation in some remote part of my hand or arm. But, if the pressure was sufficient to excite the deep system of end-organs, I showed a tendency to localize the stimulus in the neighbourhood of the spot where it was applied. The ultimate results depended to a great extent on the relative strength of the two sensations; the dominant reaction tended to determine my localization of the site of the stimulated spot.

At first sight it is difficult to understand the significance of this double method of projection; but, in the diffuse response of the protopathic system, we find the remains of an old protective reaction, intended to preserve the part from injury. A reaction of this kind is admirably fitted to defend the animal from noxious influences; it produces movements of withdrawal, which permit of no choice. It is essentially segmental, and is seen in its most perfect form in the distribution of referred pain and tenderness due to visceral disease. The internal organs are supplied from the protopathic system; there is no controlling epicritic mechanism. Any stimulus capable of exciting pain produces a segmental response; the sensation radiates widely and is referred to remote parts, including the surface of the body. Whenever the site of the stimulated spot can be correctly indicated in visceral disease, this is due to the coincident excitation of end-organs of the deep afferent system, present in the coverings and tissues of the viscera.

With the higher development of function in the central nervous system these segmental responses are replaced by massive reactions of withdrawal affecting the body as a whole. Local defensive retraction is replaced by movements of the whole animal. The mass-reflex, which occurs in certain favourable cases after total division of the spinal cord, is an example of such a method of response. The lightest stimulation of the sole of the foot may still evoke some small adapted movement; but a vigorous scratch, or a series of pricks, causes a violent flexion of the lower extremity at all joints and contraction of the abdominal muscles. This may be associated with increased activity of the bladder and rectum and an outburst of sweating in the parts supplied from the cord below the lesion. All local adaptation to the site or nature of the stimulus is swept away in a violent and indiscriminate outburst of energy in centres cut off from higher control.

Both the segmental and the massive response are means of defence and lead to withdrawal of the part from noxious influences. But they hamper voluntary action by the uncontrolled movements they evoke, and tend to prevent escape by fixing the body in a position unfavourable for flight. The animal crawls into a hole to die or to recover, whilst man becomes fixed to his bed. It is necessary, therefore, that these modes of reaction should be brought under control of centres which allow of choice and determine movements of the complete animal. The chief of these centres is the cerebral cortex which, as we have shown, is devoted to the more highly projected aspects of sensation. It is by means of sensory projection that the affective reactions can be controlled. When we attempt to climb through barbed wire, we are forced to respond not only to the pain but to the position of the wire. The pain produced by pricking a protopathic hand is all-compelling; it is impossible not to make a movement of withdrawal. But under normal conditions this ungovernable reaction is controlled by the existence of those forms of sensibility which underlie recognition of relations in space. This enables us to choose whether the hand shall be removed or not.

Thus, in their projectional characteristics, the defensive reactions in man still show evidence of stages in their functional development. The lowest form of response is segmental, seen in the case of referred pain and tenderness of visceral origin. Higher in the scale stands the mass-reflex, which may make its appearance after injury of the spinal cord. The situation of the stimulus does not determine the distribution of the response; local signature is abolished, and the outburst of energy overflows into channels that would be blocked under normal conditions. This inevitable and diffuse reaction is in turn brought under control by those centres which are concerned with postural adaptation. They determine whether a stimulus shall be followed by withdrawal of the affected part or by locomotion of the animal as a whole. Thus, it is essentially the spacial elements in sensory impressions which have led to the transformation of an inevitable segmental reaction into a discriminative response of the complete organism.

It is possible to recognize the qualitative aspects of a sensation without attributing them to an external stimulus; such reactions are related to ourselves, and do not of necessity reveal the character of the stimulating object as a constituent of the world around. Affective states associated with these sensory qualities are discontinuous; for pleasure and discomfort can no more form parts of a sensory continuum than pain, heat, and cold. They appear and disappear in consciousness, toning the sensation in a distinctive manner, without forming a connective factor in the activities of the mind.

On the other hand, the projected aspects of sensation are not related to ourselves, but to external objects. In fact, an "object" might be defined as a complex of projected responses; it is said to have characters, such as size, shape, weight, and position in space, which distinguish it from all others. The recognition of such features, however, depends on physiological activities, the product of certain definite centres in the cortex. If these processes are unable to influence consciousness, the "object" disappears, although its affective and qualitative aspects still produce their appropriate sensory reactions.

These physiological responses, which are closely bound up with the activities of the sensory cortex, are characterized by a strict dependence on past events. All projected sensations leave behind them certain physiological dispositions; for instance, the existence of the schemata ensures that a movement, occurring at one moment, is measured against the consequences of those which have preceded it. This is not a psychical act but occurs on the physiological level; every recognizable change in posture enters consciousness already charged with its relation to something which has gone before, and the final product is directly perceived as a measured postural change. This is the case with all the higher projectional aspects of sensation; they form a continuous series of dispositions, determined by previous events of a like order. The unit of consciousness, as far as these factors in sensation are concerned, is not a moment of time, but a "happening." This consists of a group of occurrences belonging to profoundly different orders in the psycho-physiological hierarchy. For example, a voluntary movement of the hand to the face depends on simultaneous changes in at least three sets of serial activities. We must become conscious of the part of the face which is the goal of the intended movement. But the complete motor act cannot occur without the participation of perfect postural schemata; it also demands the existence of an appropriate state of active muscular tone. All these three factors are products of the immediate past, active at the moment when the movement is willed, and may be affected by injury to the sensory cortex.

The sensory activities of the cortex are not only responsible for projection in space, but also ensure recognition of sequence in time. For this would be impossible if a sensory impression had no necessary beginning or termination. One of the commonest defects produced by a cortical injury is this want of temporal definition; a stimulus, rhythmically repeated, "seems to be there all the time." The patient cannot appreciate the moment at which it is applied or removed. There is no complete recognition of an extended sequence of events.

Thus, it is the projected elements in sensation to which we owe our conceptions both of coherence in space and in time. I have attempted to show that these factors are not essentially due to judgment or conscious association, but depend to a great extent on physiological activities and dispositions. When these are permitted to excite consciousness, they appear as an ordered sensation, related to other events in the external world and extended serially in time.

SUMMARY.

The Method.

(1) All the tests we have used in our previous work on disturbances of sensation were chosen because they gave measurable results. But the numerical answers yielded by the one could not be directly correlated even with those of another closely related test.

(2) A lesion of the central convolutions sometimes produces loss of sensation in the hand which is not distributed uniformly over all the digits. Some of them may be grossly affected, others may show little or no sensory disturbance.

(3) The first sign that sensation is affected in consequence of a cortical lesion is the statement of the patient himself, that the tests are less easy on the abnormal digits. This is spoken of throughout this paper as an "introspective" change.

A more advanced disturbance is shown by a raising of the threshold. Perfect answers can be obtained if the task is made easier.

The next stage is shown by the absence of a threshold; increasing the strength of the stimulus does not of necessity produce a perfect series of responses.

(4) In such cases the test which reveals the most severe loss of sensibility will be affected over the largest number of digits.

(5) In any group of tests, which appeal to the same aspect of sensation, the one which offers the hardest task suffers most severely.

Thus, amongst the methods for examining the power of spacial recognition, a test in three dimensions is more affected than one requiring discrimination of two points or simply localization of the stimulated spot.

(6) Thus we are able to correlate these empirical tests and divide the loss of sensibility they reveal into qualitative groups by noting the number of digits affected and the nature of the sensory disturbance on each finger.

The Results obtained from the Study of such Sensory Dissociation.

(7) This method of investigation showed that the sensory activity of the cortex could be divided into three categories. It is not associated with crude recognition of touch, pain, heat and cold, but endows sensation with three discriminative faculties.

These are: (*a*) recognition of spacial relations; (*b*) a graduated response to stimuli of different intensity; (*c*) appreciation of similarity and difference in external objects, brought into contact with the surface of the body.

These three aspects of sensation can be distributed independently of one another in consequence of a cortical lesion [p. 106].

(8) *Spacial recognition.*

Some loss of power to recognize passive movements of the affected part forms one of the most frequent consequences of injury to the sensory cortex. In fact, it may be accepted for clinical purposes as a leading sign in the syndrome of cortical disease. This test is a means of investigating the appreciation of spacial relationships in three dimensions; the compass-test and "spot-finding" (topical localization), as we have employed them, are explorations in two- and one-dimensional recognition.

Whenever the spacial aspects of sensation are disturbed by a lesion of the cortex, the appreciation of passive movements is most extensively and most gravely affected. Discrimination of two compass-points is less severely disturbed and the loss of sensation occupies a smaller number of digits. Lastly, localization carried out strictly according to our method shows the smallest of any of the three tests.[1] [Table on p. 109.]

(9) *Intensity.*

From the cortical aspect the tests we use for measuring sensibility to touch, to temperature and to pain are not of equivalent value. The tactile hairs demand a high degree of constancy and a strictly graduated response to stimulation. Appreciation of heat and cold, as we are forced in most cases to examine it clinically, depends comparatively little on cortical activity; for whilst it is easy to say whether an object is hot or cold, it requires an intelligent patient and favourable conditions to test the relation of two warm stimuli to one another or to determine the thresholds for heat and cold with approximate accuracy. Similar difficulties occur, to an even greater degree, with all measurable tests for painful stimulation.

At the same time these three qualities of sensation are dependent on the physiological activities of the cortex and optic thalamus to a profoundly different degree. Correct appreciation of the tactile hairs demands a high grade of cortical function, whilst a prick appeals almost entirely to the optic thalamus.

Consequently, the tactile hairs show the greatest and most extensive loss as the result of a cortical lesion. Appreciation of thermal stimuli suffers to a less degree and over a smaller number of digits, whilst sensibility to measured prick is rarely affected. [Tables on pp. 114 and 115.]

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[1] This statement presupposes that the patient is able to recognize touches with some approach to uniformity. The effect of want of tactile appreciation on these tests is dealt with in Chapter IV, § 5, p. 124.

(10) *Recognition of similarity and difference.*

The tests which come under this category are those for appreciation of size, shape, weight and texture, apart from their purely tactile qualities.

As far as the cortex is concerned, these aspects of sensory discrimination tend to be disturbed altogether. On subcortical levels, each of them depends on more than one stream of afferent impulses. But as soon as we have to deal with loss of sensibility of cortical origin, we are face to face with destruction of the activities of the centre itself and not with disorders of conduction.

All these tests, the weights, the different shapes and stuffs of various textures, demand the same power of discrimination and are found to fail together. Tests capable of measurement, such as those for the appreciation of weight, will obviously reveal a slighter degree of change than those which consist in recognition of certain fixed shapes and textures.

The faculty of recognizing the similarity and difference in objects brought into contact with the body depends upon one of the fundamental functions of the sensory cortex. It can be disturbed apart from the power to appreciate spacial relations and independently of the graduated relation to stimuli of different intensity; it may also be preserved in spite of gross defects in these functions.

(11) *Tests which depend on more than one of the fundamental functions of the sensory cortex.*

(*a*) The simultaneous compass-test demands that a series of contacts can be appreciated with some reference to the intensity of the stimulus. But, granting that tactile sensibility is not affected, the compass-points are a method of testing recognition of relations in two-dimensional space. A cortical lesion not uncommonly disturbs both aspects of sensibility and it is important to determine how each form of sensory loss affects the records of the compass-points.

When this test is disturbed in consequence of defects of tactile appreciation, the answers are confused and errors appear, not only in the recognition of two points, but also when the patient has been touched with one point only [p. 128].

If, however, tactile sensibility is not affected the patient is unable to distinguish single and double contacts and shows a tendency to call them all "one" [p. 127].

(*b*) Topical localization (or "spot-finding"), as carried out by our method, depends on the due appreciation of relative intensity and is also one aspect of spacial recognition.

Observations in selected cases seem to show that a defective response to contact stimuli appears on the records as a diffuse localization. The patient may pass his indicating finger up and down the assistant's finger, saying, "The spot seems spread out so that I can't exactly say what part of it you have touched" [p. 131].

When, however, contacts are appreciated perfectly but recognition of spacial relations is disturbed, the loss of localization may take another form. The patient is unable to decide which digit has been touched; he complains, "I have difficulty in making up my mind." The error takes the form of inability to determine on which finger or on what part of it the touch has fallen [p. 129].

(*c*) The power of recognizing various stuffs by feeling them with the fingers is obviously dependent on the accuracy of tactile impressions. But there is another aspect of this test which demands the essential faculty of appreciating similarity and difference.

By choosing suitable cases this appeal to two sides of cortical activity can be demonstrated. When the orderly response to contact of graduated intensity is disturbed, the patient hesitates and is puzzled by the curious sensory differences in his two hands; he may, however, name the stuffs more or less correctly (p. 133). If the power of appreciating similarity and difference is alone affected, his answer approximates more or less to "I don't know what it is," and he cannot name correctly the various textile substances (p. 132).

(12) *Tests which appeal to centres in the cortex and in the optic thalamus.*

(*a*) *Measured prick.*—The sensory effect produced by a prick owes something to the physiological activity of the cortex; but this is small compared with the overwhelming part played by the optic thalamus.

A cortical lesion produces no true raising of the threshold to prick unless it is extensive or is associated with subcortical destruction. It may, however, induce a change in the sensation produced by pricking, which is recognized by the patient. The two hands "feel different"; it is "plainer," "sharper," "more distinct," over the normal than over the affected digits. Sometimes the whole character of the sensation is said to be changed, and seems to "tingle" or to resemble "electricity."

(*b*) *Vibration* (*tuning-fork test*).—Disease or injury at lower levels of the nervous system may destroy all power of appreciating vibration. But so grave a loss never occurs in consequence of a stationary lesion of the cortex unless it happens to be associated with epileptiform attacks or other causes of shock; for vibration appeals both to the optic thalamus and to the cortex, although the latter element is the more important in the ultimate sensation.

A tuning-fork, beating 128 vibrations in the second, is almost always appreciated for a shorter period over the parts affected by a lesion of the sensory cortex. This loss of appreciation runs *pari passu* with the want of recognition of passive movement [Table, p. 145]. From the peripheral end-organs to the cortical centres both tests appeal to the same physiological functions.

Subcortical Lesions.

(13) The nearer the lesion lies to the internal capsule, the more extensive is usually the loss of sensation, and the more certainly does it tend to assume a hemiplegic distribution. Moreover, the sensory disturbance is extremely gross compared with that due to more superficial lesions [p. 134].

The closer a subcortical injury lies to the surface of the brain, the more nearly does the character of the sensory disturbance approximate to that produced by lesions of the grey matter. But the grouping of the dissociated sensibility does not correspond to the three cortical categories, recognition of spacial relations, of graduated intensity and of the similarity or differences of the stimulating object. The loss of sensation still reveals the arrangement of afferent impulses before they have been subjected to integration by the sensory cortex [p. 136].

Hypotonia.

(14) An uncomplicated lesion of the sensory cortex may produce hypotonia. This is closely associated in distribution and degree with the want of recognition of posture and passive movement (Tables on pp. 152 and 154]. Both defects are due to a disturbance of those physiological dispositions in the cortical centres which have been called schemata.

(15) This is a completely different condition from paralytic hypotonia, which is associated with gross loss of voluntary motor power [pp. 155 and 156].

Anatomical Localization of these Sensory Functions.

(16) Loss of sensation of the cortical type may be produced by a lesion of the pre- and post-central convolutions, the anterior part of the superior parietal lobule, and the angular gyri. These portions of the hemisphere contain the sensory centres.

(17) The form assumed by a cortical loss of sensation is not expressed in anatomical or physiological terms, but in those of the most elementary processes of mind. The anatomical lesion upsets the orderly sequence of physiological processes, and this defect of function is manifested in disturbance of the psychical act of sensation.

(18) Changes in sensibility of cortical origin on the upper extremity do not follow the distribution of axial or radicular lines. Each digit is a unit, which can be used in an isolated act either of motion or sensation; as such it is represented topographically on the cortex. The same rule applies to the palm of the hand, where the changes in sensibility correspond to the proximal extension of the affected digits. The elbow and shoulder are separately represented, because each of these joints is an important centre of the postural activities of the upper extremity.

(19) It is the functions, rather than the anatomical relations, of any one part of the body that are represented. Consequently those portions, such as the hand, which are endowed with the highest powers of discriminative sensibility, are most exclusively represented. Next in order comes the sole of the foot, which constantly exerts a discriminative action in walking.

Hence a cortical lesion may disturb the sensibility of the hand and foot without of necessity affecting the elbow, shoulder or knee.

(20) If the loss of sensation is greatest over the little finger and its neighbouring digits, the foot tends to show some sensory or motor disturbance. But when the loss of sensation reaches its maximum over the thumb and index finger, the face and tongue are liable to be affected; if the lesion lies in the left hemisphere, speech will probably have been disturbed, to a greater or less extent, at some time in the history of the case. [Tables on p. 168.]

(21) The three aspects of sensibility, due to the activity of the cortex, are not disturbed uniformly in every instance of a lesion to the surface of the hemisphere. In certain rare cases complete dissociation may occur. [Table on p. 174.]

(22) This shows that the three fundamental functions of the sensory cortex are not represented to an equal degree in all parts of its extent.

(*a*) Injury anywhere within the sensory area on the hemisphere is liable to cause some loss of appreciation of spacial relationships in the hand or in the foot. But the farther forward it lies within this region, the grosser and more extensive is the disturbance shown by the spacial tests.

(*b*) The power of recognizing differences in weight and shape is most severely and most extensively affected by a lesion which falls over the post-central convolution.

(*c*) Those tests which depend mainly on due recognition of stimuli of graduated intensity are more likely to be disturbed, when the injury falls upon the posterior and inferior portions of the sensory area. Any lesion lying mainly within a zone which occupies the foot of the pre- and post-central convolutions, the angular and supra-marginal gyri and the superior parietal lobule, is liable to be associated with disturbances of tactile sensibility. If this is severe and extensive, the patient may be unable to distinguish various degrees of warmth about which he has no doubt on the normal hand.

Theoretical Conclusions.

(23) The sensory disturbance produced by a cortical lesion is not a paræsthesia or a hypoæsthesia, but a definite loss of power to appreciate one or more of the three aspects of sensation, which depend on the physiological activity of the cortex.

(24) Analysis of afferent impulses, on their passage through the central nervous system, tells us nothing of their origin or final fate. If we wish to learn how they arose, we must produce an analogous dissociation in the peripheral nervous system; and their ultimate grouping can only be shown by breaking up those complex physiological activities which directly underlie sensation. At either end of the chain we are brought face to face with elementary physico-physiological and psycho-physiological processes respectively.

(25) If the functions of a lower more primitive organization are kept under control by the activity of a higher afferent system, the removal of this dominant mechanism does not exhibit the vital processes of the phylogenetically older organs in all their primitive simplicity. The setting free of a primitive activity from higher control reveals a condition which is part of the complete act. Thus, the reactions of the human optic thalamus, freed from the restraining influence of the cortex, are an almost perfect expression of the non-discriminative aspects of sensation.

(26) When dissociation occurs, either on the physiological or psychical level, the manifestations represent that portion of the complete function which still remains active.

Should the function lost belong to the same level as that which remains, the loss of the one does not materially affect the other e.g., dissociation of the various aspects of cortical sensibility). If, however, the factor which has been eliminated exercised some control over that which remains, primitive characters may appear that are not manifest under normal conditions (e.g., the thalamic syndrome).

(27) The simplest physical stimulus, acting on the peripheral nervous system, may produce afferent impulses which are incompatible from the point of view of sensation. These are sorted and re-grouped; some are facilitated, others are repressed, before the final sum is presented to consciousness. Many afferent impulses remain on the physiological level and never form the basis of a sensation; they are destined to control reflex activity or to co-ordinate movements of the body and limbs.

(28) The simplest sensory act is the result, not only of prolonged qualitative integration, but of a constant struggle for functional dominance. On the higher physiological planes, impulses which are precluded from exciting sensation are not wiped out; they may produce a profound and manifest effect, although they cannot excite consciousness.

The closer we come to those centres which directly underlie sensation, the more evident is it that impulses, which fail to excite consciousness, do not of necessity cease to exert any physiological influence. They may be repressed by their successful rivals, but they are not therefore abolished.

(29) Similar physiological processes underlie the afferent integration of a reflex and the co-ordination of two sensations or even of two concepts. But, in each instance, the product of these vital functions is given in terms of the level at which the impulses exert their influence; in the one case it is expressed reflexly, in the other it is manifested in some psychical act, such as sensation.

APPENDIX.

SHORT ACCOUNT OF SOME OF THE CASES ON WHICH THIS RESEARCH IS BASED.

This portion of the paper consists of two parts. In the first I have given a short account of a series of illustrative cases [Nos. 2, 3, 5, 6, 8, 15, 18, 19, and 23].

In the second part I have described as far as possible the situation and nature of the lesion in each case. Professor Elliot Smith has kindly localized approximately the site of the injury on the surface of the hemisphere from the data given to him. I am fully aware how difficult it is to judge of the position of a cerebral lesion from a wound on the surface of the skull, but reproduce figs. 3 to 23 with a full consciousness of the uncertainty of such localization.

(A) SHORT ACCOUNT OF A SERIES OF ILLUSTRATIVE CASES.

Case 2.—*An example of injury to the cortex without penetration of the skull. Wounded January* 26, 1916, *by the fragment of a shell in the anterior part of the left parietal region. Not trephined. Radiograph showed injury to outer but none to inner table of the skull. Examined in the third and fourth weeks after the wound. Right upper extremity only affected. Grasp of right hand diminished. Individual movements of the fingers were defective. Writing changed. No hypotonia. Right wrist-jerk increased. Other reflexes normal. Sensation most severely affected over the little finger, rapidly improving towards the pre-axial parts of the hand. Passive movement badly appreciated over four digits, the compass-test over one; localization not affected. The tactile hairs, thermal tests and prick showed introspective changes only, confined to the little finger. Weights and the texture of various fabrics were not perfectly recognized over the little finger. He greatly improved, and a set of observations made ninety-four days after the wound showed changes in the little finger only.*

Lieutenant F. W. B., aged 31.

On January 26, 1916, he was hit by the fragment of a shell which burst in the air. He fell, but does not think he lost consciousness. The right arm "dangled loosely and was all pins and needles"; he could, however, move it at the shoulder. He walked to the dressing station and, after a night spent in the field ambulance, was sent to the base next day. On February 5 he was admitted to the Empire Hospital for Officers, under my care. He thought his arm had rapidly improved up to January 31, but since that time its condition had remained stationary. Neither face nor leg had been affected at any time.

On admission, the wound consisted of a stellate bruised tear in the scalp about 2·5 cm. in length, which healed rapidly. The centre of this cut was situated 19 cm. from the nasion and 5 cm. to the left of the middle line. The total nasion-inion line measured 34 cm. A radiograph showed injury to the outer, but none to the inner table of the skull.

The following observations were made in *February*, 1916, *during the third and fourth weeks after the wound.*

He was a most intelligent man, an engineer by profession, and his introspective experiences were unusually definite and well expressed. The examination interested him and he showed no general fatigue. The effects of local fatigue were obvious in the readings from the affected parts, but elsewhere his answers remained remarkably constant from day to day.

Speech was normal. There was some headache at first, but it rapidly disappeared. There was no nausea or vomiting, even when he was first hit. He had not suffered from convulsions or seizures.

Vision was unaffected and the discs showed no change of any kind. The pupils reacted normally, ocular movements were well performed and there was no nystagmus.

Face, tongue and palate were unaffected.

Reflexes.—The only abnormality in the superficial or deep reflexes was shown by a definite increase of the right wrist-jerk compared with the left.

Motion.—The grasp of the right hand was certainly somewhat affected; for, although he was by nature right-handed, the grasps were equal. Individual movements of the digits of the right hand were less quick than those of the left, and he said, "All the fingers come forward and I have to pick out with the thumb the one I want to touch." This exactly expressed the condition observed. The greatest clumsiness occurred, when he attempted to approximate the tip of the thumb to that of the little finger. When he held out his hands in front of him, there was slight difference only in the alignment of the fingers. But when his eyes were closed the little finger became abducted and fell away, and the inco-ordination of the individual movements of the other digits was greatly increased.

There was no wasting and no change in tone.

All the movements of the right elbow and shoulder were strongly performed against resistance, but he was somewhat clumsy, when he attempted to touch his nose rapidly with his right forefinger. This was not increased by closing his eyes.

He could still throw with remarkable accuracy, but his writing was somewhat affected; this was particularly evident in the curves of such letters as B, M, N and W. He found it easier to write with a pencil than with a pen, for the nib seemed to catch in the paper.

The lower extremities were in every way normal and the right leg had never been affected.

Sensation (between February 9 and February 24, 1916).

He said he was in every way normal except in the right hand, where his little finger and corresponding parts of the palm and dorsal surface "felt stiff." At first this "feeling" occupied the ulnar aspect of the forearm to the elbow; but this had to a great extent passed away.

	Right (affected) hand					*Left hand*
	Little finger	Ring finger	Middle finger	Index finger	Thumb	Normal digits
Measured movement	12° to 25°; gross errors	5° to 8°	6° to 10°	3° to 6°	1°. Normal	1° or under
Vibration ..	Gross loss	Affected	Affected	Affected	Equal R. and L.	—
Compass-test	Perfect at 2 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.
Localization	Perfect	Perfect	Perfect	Perfect	Perfect	Perfect
Tactile hairs	14 grm./mm.^{2}; introspective change	14 grm./mm.^{2}	14 grm./mm.^{2}	14 grm./mm.^{2}	14 grm./mm.^{2}	14 grm./mm.^{2}
Thermal ..	Slight change; introspective	Normal	Normal	Normal	Normal	—
Prick ..	Slight change	Same R. and L.	Same R. and L.	Same R. and L.	Same R. and L.	Threshold, ·3 grm.
Weight ..	Slower; introspective change	Perfect	Perfect	Perfect	Perfect	Perfect (twelve tests on each finger)
Texture ..	Defective	Good	Good	Good	Good	Quick at recognizing all four fabrics[4]

The lower extremity was entirely unaffected.

Measured movement.—Of all the digits of the right hand the thumb alone was unaffected. In the index, middle and ring fingers the extent of the passive movement necessary to evoke recognition was considerably increased, but in the little finger the answers were confused; in three instances he indicated movement in the wrong direction with a range of 20° to 25°. He said, "I appreciate some movement, but cannot tell its direction with certainty."

Compass-test.—The little finger alone was affected and the threshold was raised to 1·5 cm. But even at this distance his answers were doubtful, though correct, and it was not until the two points were separated by 2 cm. that he became certain. He complained: "The thing that puzzles me on the little finger is that it seems like an echo. I don't know whether it is two or one with an echo. When they are close together I can't tell whether it is one point, which influences the part around, or two points. When they are wide apart, the second point falls outside this part influenced, and I know it is two."

Tactile hairs.—He gave a perfect series of answers on the little finger even with a hair of 14 grm./mm.^{2}, but the response was slower from the right than from the left. He said, "The first two or three touches were fairly clear; but it seemed to become more difficult and I wasn't so certain. I was doing it with an effort on the affected hand, instead of not thinking about it."

Thermal tests.—The only change was discovered in the little finger. The threshold for heat and for cold was not materially different in the two hands, but such temperatures as 30° to 40° C. seemed warmer on the normal than on the affected side.

Prick.—There was a definite introspective change between the sensation in the two little fingers; with 3 grm. he said the prick was "quite clearly felt" on the normal hand, but was "occasionally a slight prick" on the right little finger. On increasing the strength of the stimulus to 3·5 grm., he said it was "clearly a prick" on both hands. No such difference was found between the sensation from the two ring fingers.

Weight.—When various weights of between 20 and 100 grm., covering exactly the same surface, were added and removed, he gave quick and perfect answers on all but the little finger of the right hand. Here I was forced to wait longer and he responded with much less certainty. He said, "I've more difficulty with the little finger. The changes in weight are not so pronounced, and I was not so absolutely sure that I was right as on the other fingers."

This difference came out even more clearly, when he was permitted to lift the finger on which a weight had been placed. When asked to compare 80 and 100 grm. he made no mistakes anywhere except on the little finger of the right hand; here out of six tests he failed in three and was not perfect in his answers when comparing 70 and 100 grm. He said, "On the right little finger it is more of a strain; unless I pick it up at once I can't get it at all. It seems to merge into a confused feeling."

Texture.—For this test on the right hand he invariably used the thumb and index finger, and when forced to use the little finger he said he judged by the sensation of the thumb alone. When the various fabrics were rubbed across the digits, he complained that they "felt different on the right little finger." Here he recognized silk and cotton, but was uncertain about flannel and ribbed velvet, except that he said flannel was "rougher." When each fabric was rubbed over one little finger and then over the other, he failed to recognize that the ribbed velvet was the same substance on the right as on the left hand; the same error occurred with silk.

He was examined again on *April* 30, 1916, *ninety-four days after the wound.* He still noticed the same difficulty in writing, but found no difference when playing golf.

The arm-jerks were now equal. The grasp of the right hand had greatly improved, but isolated movements of the little finger were still clumsy. When he held his hands out in front of him, the various parts were in perfect alignment, and the little finger no longer fell away, when the eyes were closed. There was no hypotonia.

Sensation had improved greatly. All the digits of the right hand gave perfect readings with 1° of *passive movement*, except the right little finger, where from 1·5° to 2° were required. To the *compass-test* he had no difficulty at 0·75 cm. on all the digits, except the little finger; here he failed at this distance, but gave a perfect series at 1 cm. He said, however, "On the right little finger I still have the feeling of an echo. It is harder for me to tell whether there are two points, but directly you put them further apart I am certain." *Localization* was perfect. He could appreciate a *tactile hair* of 14 grm./mm.[2] on his right little finger, but said the sensation was "duller and fainter, less clear" than on the normal hand. *Weights* and *texture* were now perfectly appreciated.

Case 3.—*An instance of dissociation of sensory functions due to a cortical injury. Wounded June 7, 1917, by a rifle bullet. Trephined over the right Rolandic area June 8, when "a large piece of bone and extradural clot were removed." Admitted under my care July 7, 1917, with an unhealed wound in the right parietal region. At that time the lower part of the left half of the face moved poorly and the tongue was protruded to the left. Left arm-jerks were exaggerated. Left lower abdominal reflex defective. Left hand affected; individual movements impossible. Movements of left wrist and elbow less powerful than those of right. No hypotonia at this stage. Sensation showed from the first profound dissociation. The spacial aspects were not normally appreciated, and the power of recognizing similarity and difference was diminished, but he reacted perfectly to graduated tactile and thermal stimuli. He remained under observation and was examined from time to time. A series of records, made during the 10th and 12th months after the wound, showed that hypotonia had gradually appeared in those fingers in which the loss of recognition of posture and passive movement was most severe. This hypotonia accompanied the return of motor power and recovery of the reflexes. Sensation showed the same fundamental dissociation as before. This made it possible to work out the form assumed by the loss of appreciation of weight and texture, in the absence of changes in tactile sensibility. It also showed the nature of defects in the records of the compass-test, when spacial relationships are disturbed, but touch is not affected.*

Bombardier J. W. B., aged 26.

On June 7, 1917, he was hit by a sniper's bullet, which penetrated his helmet. He remembers a singing in his head and a feeling as if two hammers met; then he dropped. He noticed that his left arm was weak, but after about twenty minutes he could walk with help though his left leg was distinctly affected at first. After his wound was dressed, he walked about 5 kilometres to the motor ambulance. He arrived at Boulogne on June 8, and was trephined over the Rolandic area. The field card states that "a large piece of skull and extradural clot" were removed.

He was admitted to the London Hospital on July 7, 1917, with a deep lanceolate wound, situated from 17·5 to 19 cm. back from the nasion, and from 4 to 8 cm. to the right of the middle line. The total nasion-inion line measured 35 cm. The wound had a granulating base, which pulsated heavily. Cultures grew *Staphylococcus albus* only.

Condition in July, 1917, *forty days after the Wound.*

The importance of this case lies in the condition, which was present many months after the wound had healed; but I shall summarize first of all the observations made shortly after his first admission to hospital.

His mental state was excellent and speech had not been affected at any time.

Vision was perfect, the fields were not restricted and the discs were normal.

The pupils reacted normally, movements of the eyes were well performed and there was no nystagmus.

The lower part of the left half of his face did not move so well as the right, and the tongue was protruded a little to the left.

Reflexes.—The left wrist-jerk and triceps-jerk were distinctly exaggerated, but the knee- and ankle-jerks were equal and there was no ankle clonus. The plantar reflexes were normal. A reflex could be obtained from all four areas of the abdomen; but, whereas those from the right upper and lower segments were quick and vigorous, the reflex from the left lower quadrant was obtained with difficulty and was of small range.

Motion.—The grasp of the left hand was profoundly diminished and registered on the dynamometer 5, compared with 58, the force exerted by his right hand. He could not carry out individual movements with the fingers of the left hand; they tended to remain passive or to be flexed in a bunch, whilst the thumb moved across to be approximated to the tip of each finger in turn. Movements of the left wrist were feeble and those of the elbow were not so strong as on the other side. The shoulder was not affected.

The fingers of the left hand were not in alignment, when the hands were held out in front of him; the wrist became flexed and the whole arm swayed unsteadily. As soon as the eyes were closed the hand dropped and the fingers became irregularly flexed. Even with the eyes open, on attempting to touch his nose, the movements of the left hand were inco-ordinate and unsteady, and when they were closed the ataxy was pronounced.

There was no obvious spasticity and no hypotonia.

The left forearm and hand were distinctly wasted; this was most visible in the interossei and was less evident in the muscles of the thumb. The arm and shoulder muscles were not affected.

In the left lower extremity there was no gross loss of power and no wasting; but the movements were not quite so accurate as those of the right leg. He could, however, stand equally well on either foot with the eyes closed.

Sensation.—The outstanding feature of the sensory disturbance was the considerable want of recognition of measured movement in all the digits and the wrist, together with gross loss of discrimination of weights in the little and ring fingers, in spite of perfect sensibility to touch. This is a case where the various sensory activities of the cortex were dissociated from an early stage.

I shall, therefore, pass over his condition during the intermediate period in order to lay stress on the observations made during *March, April and May*, 1918, *ten to twelve months after the wound.*

The scar was firm and did not pulsate. He had been discharged from the Army and was at work, rarely suffering from headache except when tired.

He was remarkably bright and intelligent, handing himself over willingly for examination. His answers were given without hesitation; he made no attempt to interpret his sensations and was not introspective.

Reflexes.—Great improvement had taken place and the two elbow-jerks were almost of equal strength; there was no difference between the triceps-jerks. The abdominal reflexes were equally brisk on the two sides in all the areas.

Motion.—The grasp of the left hand had greatly improved and individual movements could now be carried out by each finger in turn. They were, however, slower and less perfect than those of the right hand, which were quick and precise. The various digits were in good alignment, when he held his hands out in front of him, but they fell away when he closed his eyes and the movements of the fingers of the left hand became distinctly ataxic.

The little and ring fingers of the left hand, and to a less degree the middle finger, were now definitely hypotonic, whilst the index and thumb were normal.

There was no wasting; but the ulnar half of the left hand was soft and toneless to the touch, compared with similar parts of the right.

Sensation.—He was at work but had no necessity to use his left hand except for lifting. He could pick up objects with the forefinger and thumb but complained that he was clumsy with the middle, ring and little fingers.

	Left (affected) hand					*Right hand*
	Thumb	Index finger	Middle finger	Ring finger	Little finger	Normal digits
Measured movement	1°. Introspective	1° to 2°. Direction defective	1° to 2°. Direction defective	Gross loss	Gross loss	Perfect at 1°
Vibration ..	Not affected	4 to 6 sec.	4 to 5 sec.	10 sec.	10 sec.	—
Compass-test	1 cm.	1 cm.	1 cm.	Gross loss at 5 cm.	Gross loss at 5 cm.	Perfect at 1 cm.
Localization	Good	Good	Good	Affected	Affected	Good
Tactile hairs	Normal	Normal	Normal	Normal	Normal	Perfect with 14 grm./mm.^{2}
Thermal ..	Normal	Normal	Normal	Normal	Normal	Can distinguish 35° and 40°C. perfectly
Prick ..	Normal	Normal	Normal	Normal	Normal	Threshold about 4 grm.
Weight ..	Perfect	Perfect	Distinctly affected	Gross loss	Gross loss	Perfect appreciation of 80 and 100 grm. Perfect answers to addition and subtraction
Form ..	Perfect	Perfect	Slightly affected	Gross loss	Gross loss	Perfect appreciation of the six tests. Objects rolled over palmar aspect of fingers
Texture ..	Perfect	Perfect	Affected	Gross loss	Gross loss	Perfect appreciation of ribbed velvet, silk, flannel and calico rubbed over digits

Passive movement was appreciated within normal limits on the left thumb, but he said it was "less easy" than on the right hand. With the index and middle fingers the readings were slightly raised, and he tended to be somewhat uncertain of the direction of the movement with an excursion of 1° to 2°. Both ring and little fingers were grossly affected; he did not appreciate a movement of less than 20° to 30° and in many instances its direction was wrongly indicated.

The *compass-test* gave normal readings over the thumb, index and middle fingers of the affected hand, but over both ring and little fingers contact with two points was invariably called "one," even when they were separated to a distance of 5 cm. He said, "I feel the pressure quite well, but it all feels the same. It is not like the other fingers."

Localization had throughout remained unaffected on the thumb. In view of the normal state of tactile sensibility, I chose the palmar aspect of the four post-axial digits for a more extended series of observations than usual on topical localization. Twelve contacts were made, first on the normal and then on the affected side; this was repeated three times on each hand to the number of seventy-two in all. Every touch on the normal hand was correctly localized, but the answers from the fingers of the affected side showed a disturbance increasing in a post-axial direction. Thus, whilst all nine stimuli were correctly localized over both the left index and middle fingers, he made two mistakes over the ring, and six over the little finger. Of these six errors one was placed on the wrong digit, three were more or less distal, and two were proximal to the spot stimulated. Both the mistakes on the ring finger consisted in choice of the wrong digit.

All the digits of the affected hand gave perfect records to the *tactile hairs*, even with 14 grm/mm.^{2}, the smallest strength of stimulus to which he responded constantly on the normal fingers. There was no introspective difference between the two hands. *Thermal sensibility* was equally good on both sides and *prick* was not affected. The whole of this aspect of cortical activity remained throughout untouched by the destructive effects of the lesion.

But the appreciation of relative weights, form and texture was gravely affected in the little, ring and to a less degree in the middle fingers of the left hand. In view of the perfect reaction to varying intensities of stimulation, this was a most instructive dissociation.

Weight.—This case also illustrated the rule that with a cortical lesion inability to discriminate weights is independent of the manner in which they are applied. If the hand was carefully supported, and weights were added or subtracted on each digit in turn, he failed to recognize the direction in which the load had been altered in spite of perfect tactile sensibility. He was also unable to recognize the difference between two weights placed one after the other on the affected fingers, even when he was permitted to move them and to "weigh" each test-object in turn.

Thus, with the method of addition and subtraction, the thumb and index gave normal readings; on the left middle finger he did not reply to five of the twelve changes in weight, on the ring finger he did not answer on six occasions and thought all the weights had been removed, when one of 20 grm. still remained on the hand. On the little finger he failed to appreciate ten out of twelve changes and asserted that there was no weight when 140 grm. still remained on the hand. He also experienced hallucinatory changes of weight, describing addition and removal, when the pile remained untouched. He stated, "The only ones that seem to me bad are the middle, ring and little fingers; the little finger is the worst. The first (index) is all right."

The following observations were made on his power of comparing two weights by "weighing" them; they were placed one upon another, in irregular sequence, on the palmar aspect of the terminal phalanx of the little and ring fingers and of the middle and index fingers. Two digits were chosen so that the movements should not displace the weight, as would have been the case if one finger only had been selected. On the normal hand, and with the left index and middle finger he recognized the difference between 80 and 100 grm. on every occasion. But with the left little and ring fingers he could not even appreciate a difference of 100 and 20 grm. All his answers took the form of "I can't tell, I don't know," or some similar expression of ignorance.

An unusually extensive series of observations were made on his power of recognizing *form*, when solid test-objects were rolled over the palmar aspect of each digit in turn; for on the normal hand he was particularly good in recognizing the shape of the solid blocks cut to scale. On the left thumb and index he made no mistakes, on the middle he was a little uncertain, but on the ring and little fingers his answers were gravely defective. Thus on the latter he said, "Everything feels alike; except that they scratch or hang on to the flesh; I cannot tell they have any shape at all." Pyramid, ovoid and cube were all said to be "square," whilst a cylinder and cone were called "round" and a sphere was not recognized at all.

The power of appreciating differences in *texture* was gravely affected in the little and ring and, to a less degree, in the middle fingers of the left hand. The ribbed velvet, silk, cotton and flannel were named correctly over all the digits of the normal hand and over the left thumb and index. But on the middle finger velvet was called "silk," flannel was thought to be "cotton," whilst silk and cotton were named correctly. Over the little and ring fingers he either said the fabric was "rough" or confessed he did not know. "Everything seems rough on these fingers and I find it difficult to tell on the middle finger. The first finger and thumb are all right."

But in spite of this profound disturbance, his power of appreciating *roughness*, measured with the Graham Brown apparatus, was identical on the digits of the two hands.

Case 5.—To illustrate the introspective aspects of the sensory loss of cortical origin. Wounded June 6, 1916, by a rifle bullet. A radiograph showed injury to the outer plate of the skull in the left parietal region, but none to the inner surface. He was not trephined. Admitted to the Empire Hospital December 4, 1916. The wound was represented by a fully-healed scar, 3 cm. in length, in the left parietal region, 23 cm. from the nasion and 5 cm. from *the middle line. The importance of this case lay in the patient's remarkable power of describing his sensations over abnormal parts. Individual movements of the right hand were good with the eyes open, but clumsy when they were closed. The right thumb and index finger were hypotonic. In the right foot he had lost, to a certain extent, the extraordinary powers of moving his toes, which he still possessed on the normal side. Otherwise it would not have been possible to discover any affection of motor power in the lower extremity. The reflexes were normal. Sensation was most severely affected in the thumb and index finger of the right hand. In the little finger many aspects of sensation showed introspective changes only. The tactile hairs, thermal tests and prick showed the severest and most extensive loss. Sensory changes, similar in quality, but somewhat less severe, were present in the right foot.*

2nd Lieutenant R. S. C., aged 28.

In the early hours of June 6, 1916, he was hit by a sniper's bullet. He was not wearing a helmet. He spun round and felt as if someone had stuck a boathook into his head, pulling him backwards. Apparently he fell, but did not lose his senses, and after his wound was dressed he went back to his machine-gun. But "everything seemed so slow; I seemed to have lost touch with my men. I was thinking very slowly." He had a violent headache and felt sick, but did not vomit.

He stopped one day in the Field Ambulance and on June 7 reached the Casualty Clearing Station. Here a piece of the casing of a bullet was removed from the scalp. A radiograph showed injury to the outer surface of the skull, but none to its inner aspect. He was not trephined.

The wound healed, and he was discharged on June 24 from the 3rd London General Hospital, with a scab still present on the site of the injury. He could not sleep and the noise of the gramophones made him feel sick. The next four months he spent in the country, in a most unsatisfactory condition physically. He was easily fatigued and fell into violent rages; when they came on, whether by day or by night, he would leave the house and walk up and down in the garden for hours.

During the first week of November, 1916, he was sent back to light duty. The headache returned and the attacks of "violent feelings" became more pronounced; they were followed by depression and suicidal ideas. He acted as assistant adjutant, working from 9 a.m. till 7 p.m. He rapidly became so ill that he was sent to see me by his senior medical officer, on November 30, 1916, and was admitted to the Empire Hospital for Officers on December 4. Here he rapidly improved and the following account is based on observations made during *January*, 1917, *seven months after the injury*.

The wound was then represented by a fully healed scar, 3 cm. in length, in the left parietal region. The anterior limit was 23 cm. from the nasion and 5 cm. from the middle line of the skull. The total nasion-inion line measured 35 cm. A radiograph showed that the outer plate of the skull had been damaged in the region of the injury, but the inner plate seemed to be intact. There was no evidence of a foreign body or dead bone within the cranial cavity.

He was a highly educated man of unusual intelligence. His answers to measured tests were far above the average, especially from the introspective point of view. He tended, however, to interpret his sensations and this made him slow to reply, when an affected part was being tested. He gave himself over most willingly to my examination because, in spite of his repeated complaints, no one had examined into the condition of his right hand. The fact that he had never been examined, even superficially, from a neurological point of view, was one of the most potent causes of his anger and misery. His power of attention was good, but he became tired easily and care had to be taken to make the examination in short spells, selecting days when headache was absent and he had slept well. Sleep soon improved and he ceased to dream unpleasantly. His memory was not good on admission, but became much stronger with rest and quiet. There were no hallucinations or delusions at any time. At first he was irritable, but this passed away and he became particularly easy to deal with.

On admission he had a stutter which slowly disappeared; this was a purely psychical manifestation and speech was not otherwise affected. He had had no seizures or convulsions. The headache was at times severe and was of the same character throughout; it was "like a toothache," mainly on the left half of the head. All nausea had disappeared for some months before admission.

The discs were unaffected. The pupils reacted normally. All movements of the eyes, face, and tongue were carried out well.

Reflexes.—All reflexes, superficial and deep, were equal on the two sides and were normal in character.

Motion.—The grasp of the right hand was certainly less powerful than that of the left, but there was no paralysis. Individual movements of the fingers could be carried out easily so long as his eyes were open, but became very clumsy when they were closed. He could not write so easily as before he was wounded, especially if he was tired, and his handwriting had changed somewhat in character. He could still play the piano, but his right hand "was not so good as it might be."

The right thumb and index finger were definitely hypotonic; the condition of the middle and ring fingers was more doubtful, but the tone of the little finger was certainly not diminished. There was no wasting of the hand.

The wrist, elbow, and shoulder were unaffected.

Had he been brought up under ordinary conditions, there would have been no evidence that the movements of the right lower extremity were in any way affected. But as a child he had lived on a sandy beach in China and his father had not permitted him to wear boots. His agility with the toes of the normal foot was remarkable; he could cross the great toe over the second, could spread the toes and bring them together again, just as we can adduct or abduct our fingers. He had no difficulty in picking up a penny from the bed with his foot. But, with the right foot, he could no longer cross the toes, though he could abduct or adduct them, less perfectly than with the other foot. Individual movements of the right great toe were still possible, but were slower and less accurate than those of the left. There was no gross hypotonia, but it seemed to me as if the right great toe could be passively extended to a greater degree than the left, and he said, "I think the great toe of the right foot is slack like the first finger (index) of the right hand."

Apart from this loss of special aptitude in the foot, there was no want of power anywhere in the right lower extremity.

His gait was normal and he could stand well on either foot with the eyes open; but when they were closed he said, "I don't trust myself so much on the right; I have to plant it more definitely before I begin; whereas I don't mind how I put the left foot before I close my eyes."

Sensation.—He complained mainly of the index finger of the right hand and said, "If I wanted to feel for a pin I should use the first finger of the left hand, but with the right I should use the middle finger, because the first finger felt as if there was a corn on it."

	Right (affected) hand					*Left hand*
	Little finger	Ring finger	Middle finger	Index finger	Thumb	Normal digits
Measured movement	1°	8° to 15°	10° to 15°	Gross loss	Gross loss	Perfect with 1°
Vibration ..	No shortening	Affected	Affected	Affected	Affected	—
Compass-test	1 cm., introspective	Not quite perfect at 3 cm	Not quite perfect at 3 cm.	Gross loss	Gross loss	Perfect at 0·75 cm.
Localization	Good	Good	One error	Gross loss	Gross loss	Perfect
Tactile hairs	23 grm./mm.^{2}	23 grm./mm.^{2}	23 grm./mm.^{2}	No threshold	No threshold	21 grm./mm.^{2}
Thermal ..	Introspective	Affected	Affected	Gross loss	Not tested	35° and 40°, perfectly discriminated. Thresholds 30° C. and 27° C.
Prick ..	4 grm.	8 grm.	8 grm.	8 grm.	Not tested	Threshold, 4 grm.
Weight ..	Definite loss	Definite loss	Gross loss	Gross loss	Gross loss	Perfect series on addition and subtraction
Texture ..	Introspective	Gross loss	Gross loss	Gross loss	Gross loss	Perfect appreciation of four fabrics on each digit

Measured movement.—The loss of appreciation of passive movement was so gross in the thumb and index finger of the right hand that 40° sometimes evoked no reply. He said, "It is pure guessing; I have no idea which way it is moving." With the middle and ring fingers he not only recognized the movement, but also its direction at a range of between about 8° to 15°, whilst with the little finger he answered correctly in every instance to a movement of 1°.

Vibration exactly bore out the results obtained with the test for passive movement; the little finger was entirely unaffected, whilst the other four digits of the right hand showed considerable shortening of the period during which the tuning-fork was appreciated.

Compass-test.—Over the thumb and index finger, he failed entirely to recognize the two points at a distance of 5 cm., and frequently thought that contact with one point was "two." In fact, his answers on these digits showed complete confusion. On the middle and ring fingers his answers at 2 cm. were confused, but at 3 cm. they were almost perfect. On the little finger he could discriminate one and two points as 1 cm., and made no mistakes; the sensation was not, however, the same as on the normal little finger. There is no doubt that on this digit, at any rate, the compass-test was disturbed by the changes in tactile sensibility.

Localization was profoundly disturbed on the thumb and index fingers. A single contact might appear to be two, or even three touches, each of which was localized in a different position. He would call out, "Just wait a minute, I want to touch a dozen different places." Then after a pause, "I think it must have been there," indicating a wrong spot. He also said, "Unless I answer at once I am done; I go on wandering about." Now, there can be little doubt that this is an example of a double defect; he had not only lost, to a great extent, his power of appreciating the locality of the stimulus, but the sensation produced by the contact was in itself disturbed. On the middle finger he made one mistake, referring the touch to a proximal point on the ring finger. On the ring and little fingers he made no mistakes.

The greatest disturbance in this case was shown by those tests which demanded an accurate response to stimuli of different intensities. For this aspect of sensibility was certainly affected over all five digits, whilst on the spacial side even measured movement showed no defect in the little finger. Moreover, his own statements with regard to his tactile and thermal sensations throw considerable light on the nature of the changes caused by a cortical lesion.

Tactile hairs.—The thumb and index finger were profoundly affected to this test. No threshold could be obtained, and on first testing with a hair of 23 grm./mm.^{2} he gave as many correct answers as to a series of subsequent contacts with one of 100 grm./mm.^{2} (*vide* p. 117). The patient said: "On the left (normal) side I felt as if you were putting on and taking off a fine wire point. On the right hand it was exactly as if a mosquito drew its leg along the surface. I never felt a distinct pressing on. Sometimes I felt it vaguely as if it was still there, and I wanted you to press it and take it away again, so that I might be certain if it was there or not."

On the middle and ring fingers a threshold was obtained with a hair of 23 grm./mm.^{2}, but his answers were slow and he said that he had difficulty in making up his mind. On the little finger the record with 21 grm./mm.^{2} was defective, but he gave perfect replies with 23 grm./mm.^{2} There was, however, a profound introspective difference between the two little fingers; "Sometimes it seems dead and sometimes it is like the brushing of a fly's wing." Throughout these tests, he complained that the serial contacts with the tactile hairs, over affected parts only, set him "on edge." "I feel that, if you went on long, I should become like I am when I am worked up; I seem to want a relief somewhere."

Thermal tests.—The sensibility of the index finger was profoundly affected to all the tests for appreciation of temperature. He could not discriminate heat or cold with certainty between about 26° C. and 40° C., whereas on a bright spring-like day, the temperature of the room being 18° C., the indeterminate zone lay between about 26° C. and 30° C., over the normal index finger. But an even more striking defect was shown by his inability to distinguish two stimuli of the same thermal quality, but of different intensity. So gross was this change that he was unable to discriminate 35° C. and 40° C., 35° C. and 45° C., and even 20° and 25° C. (*vide* p. 218).

When tested with the warm and cold tubes he said: "There is a great difference between the two fingers; the right never changes its mind, whereas with the left (normal) the temperature seems to grow upon it; with heat I feel it growing hotter until it reaches a certain stage and then stops. Cold does the same thing. I was baffled on the right hand; I don't know how to describe it. It sometimes seems ice-cold and sometimes nothing at all; but there was never any difference between the two tubes." The cold tubes were first applied to the affected hand; as soon as the change was made to the normal hand he burst into laughter, saying: "I could not have believed that the difference could be so great. Here I never have a moment's doubt; the first was just off tepid and the second was colder still, or vice versa, as the case might be."

Over the middle and ring fingers the disturbance of thermal sensibility was less severe. He could not discriminate 35° and 40° C. on the affected hand, but there was no such profound difference between the thresholds as on the index finger.

Over the little finger he gave correct answers in every case, when 35° C. and 40° C. were compared, but said that the sensation from the affected hand was "different; on the right hand (affected) it has a dryer feeling, whilst on the normal one it is more liquid." He added: "It does not change on the right as it does on the left (normal); on the left it leaves a definite feeling, which I can think about, a sort of glow, which I feel going off."

Prick.—He complained that a prick did not seem normal on any of the digits of the right hand. The threshold was distinctly raised (4 grm. left, 8 grm. right) on the index, middle and ring fingers. On the right little finger it was 4 grm., the same as on the normal hand, but he said he "felt it more on the left; it seemed softer on the bad hand."

Weight.—Over the thumb and index finger the addition and subtraction of weights was not appreciated; he either failed to reply or his answers were confused. He complained, "Sometimes it feels as if you were rolling my finger round, not as if you were putting things on and taking them off."

Over the middle and ring fingers the loss of sensibility was less pronounced. In a sequence of twelve changes in weight he failed to answer five times on the middle and three times on the ring finger, but there was no confusion.

Over the little finger the sensation was also measurably affected and he said: "I could distinguish when you were putting on and taking off the heavier weights; but there were times when I could not tell what you were doing, it seemed as if you were only touching me."

Texture.—He was unable to distinguish ribbed velvet, silk and flannel over the thumb, index, middle and ring fingers. Over the first three digits the loss was absolute and he said he had "no idea" what I was rubbing over his fingers. On the ring finger he could not determine which of the three fabrics he was feeling. but they seemed different from one another; at the same time they all produced a different sensation from anything he experienced on the normal hand. On the affected little finger all three fabrics were named correctly, but he said there was "a decided difference between the two hands." "When you put the silk on to the left (normal) little finger it comes down softly and I feel it at once. On the right hand it might be anything, until I worked at it to get the definite silk feeling, or until you rubbed it in. With the ribbed velvet, I recognized at once on the right (affected) little finger, that it was a heavier, warmer material; but I could not make out what it was until I thought about it."

Sensation was distinctly affected on the right lower extremity, though to a less degree than on the hand.

Measured movement.—At the normal great toe, ankle and knee, he responded perfectly with passive movements of 1°. But, on the affected side, he required a range of from 3° to 6°, and from 3° to 5° at the great toe and ankle respectively before an answer was evoked. At the knee he appreciated a movement of from 1° to 2°, but he was somewhat slower than on the normal side.

Compass-test.—On the normal sole he gave a perfect set of readings at 3 cm., whilst on the right foot he was equally confused at 3, 4, 6 and 10 cm. This confusion was evidently in part due to irregular tactile responses, for he complained: "On the right foot some touches are much duller, some much more lively. Even when they seem two, the two points are quite different in feeling."

Tactile hairs.—On the normal sole he reacted perfectly to a hair of 23 grm./mm.^{2}, and to 13 out of 16 consecutive contacts with one of 21 grm./mm.^{2} But on the right sole no threshold could be obtained, even with 100 grm./mm.^{2} During the testing on the affected foot he said: "It seems as if it was on all the time. It's just the difference between a point and a straight line; on the good foot, it has position but no magnitude, on the other one (right) it has magnitude without position. I feel you must press on the part or I could not bear it. It sets me on edge, just as your teeth feel when a knife is scraped on a plate."

Thermal.—The feet are not usually a favourable field for observations on thermal sensibility, especially in the winter, and this case was no exception to the rule. However, he was certain that the sensation he obtained from the two feet was not identical with the same hot or cold stimulus, and he was unable to distinguish 35° and 45° C. on the right sole: they seemed to him the same throughout a series of observations, although they were easily recognized and named correctly when applied to the left sole.

A further set of observations were made in June, 1917, exactly twelve months after the injury, and yielded results which did not differ materially from those detailed above.

Case 6.—*An example of the effects of a subcortical injury. Wounded July* 3, 1916, *by a shell fragment in the left frontal region. Trephined July* 4. *Came under my care July* 17. *The observations recorded below were made during March,* 1918, *twenty months after he was injured. The wound lay in front of the Rolandic area, but had produced subcortical changes in motion and sensation. These affected the right hand and foot. Isolated movements of the fingers good with eyes open. No hypotonia. Movements of the toes and dorsiflexion at the ankle were less perfectly performed right than left. Right abdominal reflex diminished; other reflexes normal. Sensation was affected in the right hand, and the loss lay mainly over the post-axial parts. The records showed the raised threshold and the absence of irregular answers and hallucinations, characteristic of a subcortical lesion*

Captain R. D. D. C., aged 41.

On July 3, 1916, he was hit in the frontal region by a fragment of a high-explosive shell, which penetrated his helmet. He became unconscious for a few minutes, and then went on with his work. Shortly afterwards he "fainted," but crawled back to the dressing station. The wound was thought to have injured the scalp only, and he was passed on rapidly to Boulogne. Here he was operated on at 7 a.m. on July 4. On July 17 he was evacuated to England, and was for a short time in the Empire Hospital for Officers.

The wound healed before the end of July. It consisted of a surgical flap in the frontal region, bounded by a semi-lunar incision posteriorly, which extended further to the left than to the right of the middle line. This covered an irregular trephine opening in the skull, which began 6·5 cm. from the nasion and extended backwards for 5·5 cm. This opening, which was roughly triangular with the apex posteriorly, extended 2·5 cm. to the left and 1·25 cm. to the right of the middle line. It pulsated heavily, especially when he put his head down. The total distance along the nasion-inion line was 34 cm.

He returned to work in March, 1917, but happened to come under my care again in March, 1918, twenty months after the date of the wound. He was then in an excellent condition for detailed examination, and the following observations were made between *March* 6 *and April* 3, 1918.

The site of the wound now consisted of a deeply depressed area over the opening in the skull, which bulged when he coughed, or when he put his head down towards the ground.

His mental state was extremely good; he was a highly intelligent soldier, who had become tired in consequence of too much irritating routine under an unsympathetic commanding officer. Headaches were at first troublesome, but rapidly improved under quiet and rest. He had not suffered from convulsions or seizures of any kind.

Vision was good, and the discs showed no signs of having been affected at any time. The pupils reacted well, ocular movements were normal, and there was no nystagmus.

The movements of the face, tongue and palate were carried out perfectly.

Reflexes.—All the deep reflexes, both of the upper and lower extremity, were equal on the two halves of the body. The plantar reflexes were brisk and the great toes gave a downward response. The only reflex abnormality was distinct diminution of the response from the right half of the abdomen; in both the upper and lower segments the movement was slow, of small extent, and confined to the neighbourhood of the stimulated spot, whereas on the left side a scratch either above or below the umbilicus tended to produce a brisk movement of the whole abdomen.

Motion.—Isolated movements of the fingers and thumb of the affected hand were well performed so long as his eyes were open, but when they were closed the right little, ring and middle fingers moved clumsily and even the index did not move as accurately as the left. He complained that the "little and ring fingers are the worst, the middle finger is somewhat better, but even the first finger is a little difficult." The grasp of the right hand was obviously less than that of the left; and movements of the right wrist were also slightly weaker, but those of the elbows and shoulders were equally strong on the two sides. He used to be a strongly right-handed man, but was now using the left hand for picking up small objects or for turning the handle of a door. There was no wasting and no hypotonia.

Under normal conditions he walked well; but the right foot easily became tired, and, when this happened, he noticed that he did not "clear things" on the ground so easily as with the normal foot. When he got out of bed at night, or was walking in the dark, he always used his left foot to "feel the ground."

He could make all movements with the right lower extremity; but flexion and extension of the toes were somewhat slower, and dorsiflexion and plantar extension of the foot less strongly performed on the right than on the left side.

With the eyes open he could stand on either foot, but staggered when he attempted to balance himself on the right with his eyes closed. In testing his co-ordination, the right foot was certainly clumsier than the left; when his eyes were closed, he had distinctly more difficulty in finding the right great toe with the left heel than *vice versa.*

There was no spasticity and no hypotonia.

Sensation (between *March* 9 *and March* 30, 1918, *twenty months after the wound*).

He complained that "the three fingers (little, ring, middle) of the right hand seem as if they were always going to sleep. I have to move them about to make them come to again. When I wake at night I sometimes feel as if I hadn't got these three fingers, and the whole of that part of the hand seems to disappear, too."

I have placed on the following table the results obtained with the ten tests. They are so clear and definite that I shall only comment shortly on each set in turn.

	Right affected hand					*Left hand*
	Little finger	Ring finger	Middle finger	Index finger	Thumb	Normal digits
leasured movement	Average 19°	Average 14·6°	Average 9°	Average 8°	Average 2·5°	Perfect at 1° to 2°
ibration ..	29 sec.	27 sec.	23 sec.	28 sec.	18 sec.	—
ompass-test	1 cm.	1 cm.	1 cm.	0·5cm. Slower	0·5. Introspective	Perfect at 0·5 cm.
ocalization	Not affected	Not affected	Not affected	Not affected	Not affected	No mistakes
actile hairs	Not perfect at 110grm./mm.^{2}	Threshold 110grm./mm.^{2}	Threshold 70grm./mm.^{2}	Threshold 85grm./mm.^{2}	Threshold 85grm./mm.^{2}	Perfect reading with 14 grm./mm^2
hermal ..	No differentiation	3 errors	2 errors	Introspective	Perfect	35° to 43° perfectly recognized
rick ..	9 grm.	7 grm.	7 grm.	6 grm.	3 grm.	3 grm. thumb, 5 grm. index, 4 grm. other digits
eight ..	8 errors	5 errors	"Much more difficult"	2 errors	Perfect	Perfect series of 12 tests on each digit
orm ..	Defective	Defective	Introspective	Good	Good	Perfect recognition
exture ..	3 errors, much doubt	3 errors	Introspective	Introspective	Introspective	Named all 4 tests correctly on all digits

Measured Movement.—On each finger I have given the average of ten movements. In many cases, with a purely cortical or combined cortical and subcortical lesion, this is not possible, because of the uncertainty of the answers and of hallucinations of movement. In this case, however, among the fifty answers from the affected fingers, the direction was wrongly given in two instances only, and the figures fell within remarkably close limits on any one finger.

Vibration.—The difference between any two digits was considerable, even on the two thumbs. This test did not reveal the same fine differences of appreciation as measured movement, although the same number of fingers were affected.

Simultaneous compass-test.—He gave perfect records on the normal digits, when the points were separated by 0·5 cm. only. He was conscious of a difference between all the digits of the right hand and those of the left. But the first obvious change appeared in the index finger, where the answers were slow, although the record was perfect as 0·5 cm. Over the middle, ring and little fingers it was necessary to separate the points to 1 cm. before he could give a perfect series of replies, and the little finger was evidently most severely affected.

Localization was not obviously disturbed.

Tactile hairs.—This test revealed a condition different from that usually found with a lesion of the sensory cortex. The lighter hairs were not appreciated at all, and each succeeding increase in the strength of the stimulus gave a corresponding increase in the number of replies. The little finger was the only digit where a threshold was not ultimately obtained. There were no hallucinations or gross irregularities of response [p. 138].

Thermal tests.—When two silver test-tubes, containing water at 35° C. and at 43° C., were applied in irregular sequence to the palmar aspect of the terminal phalanges of the normal hand, he answered quickly and correctly in every case. On the right thumb and index finger he made no mistakes; he said that it was less easy over the right index than over the left. Over the middle finger he twice thought that the tubes were of the same temperature, whilst on the ring finger three out of four applications were thought to be the same. On the right little finger he failed altogether to recognize that the tubes were of a different temperature.

When 40° C. was applied to each digit of the two hands in turn, he noticed no difference between the sensation in the two thumbs; but it seemed "easier" on the normal index, and over the left middle, ring and little fingers the same tube seemed "hotter" than on the affected hand.

Prick.—To this last there was a distinct raising of the threshold over the middle, ring and little fingers of the right hand.

Weight.—This test was carried out by adding and subtracting weight on each digit in turn. From the thumbs he gave perfect answers, but on the right index he twice failed to reply, when a weight was removed. This is one of the earliest signs by which a disturbance of this aspect of sensibility is revealed in cases of cerebral lesion. Over the right middle finger he made no actual mistake, but was much slower even than over the index, and complained that he found the test "much more difficult" on the right hand than on the left. The ring and little fingers were more obviously affected, and here he failed five and eight times respectively out of twelve tests on each digit.

Form.—It was very difficult to be certain of the results of this test, when the sphere, cylinder, pyramid, cube, cone, and ovoid were rolled over each finger in turn; but he was obviously much slower in his answers from the little and ring finger of the right hand. With the middle finger he made no mistakes, but found it easier to be certain on the left than on the right hand. There was no definite difference between the thumb and index fingers on the two sides.

Texture.—Velvet, silk, cotton, and flannel were named in every case correctly, when rubbed over the normal digits. On the right hand, however, even the thumb seemed to him to be affected, although the only mistakes were found when the tests were applied to the ring and little fingers. Here he either said he "did not know" or spoke of the roughness or smoothness only of each textile fabric.

Case 8.—*This case is cited as an example of disturbed sensibility at the elbow, and to a less degree at the wrist, without obvious affection of the hand or shoulder. Wounded in the left side of the head on December* 21, 1915, *by shrapnel. He was trephined within a few hours of the injury over the middle, pre- and post-central convolutions. From the first the changes in motion and sensation lay in the neighbourhood of the elbow and wrist. The hand was slightly affected at first, but had recovered entirely six weeks after the injury.*

Lieutenant L. A. D., aged 20.

He was wounded on December 21, 1915, by shrapnel, at Cape Helles. He dropped, and was unconscious for a few seconds only.

On January 5, 1916, he was seen by Sir James Purves-Stewart, who kindly sent me the following account: "The patient immediately lost power in the right upper limb. Within an hour or two he was operated upon at the 17th Stationary Hospital, where a trephine opening was made; the anterior edge was ½ in. behind the Rolandic fissure at a point 2½ in. from the middle line, measured along the fissure. The scalp wound has completely healed. Within a couple of days the right upper limb began to recover and has since improved steadily. There has been occasional left-sided headache. No vomiting.

"His condition on January 5, 1916, was as follows: Speech and articulation were normal. Visual fields were normal and there was no hemianopsia. Pupils and cranial nerves normal. Optic discs not affected. To the lightest cotton-wool touches and to pin-pricks there was no loss of sensation in the face, trunk, or limbs, but light touches and pricks on the right forearm and hand produce a mild sensation of superadded tickling. Similarly with a vibrating tuning-fork. The joint-sense was normal at all joints. Compass-test normal on both hands (right 2 cm., left 1½ cm.) Stereognosis perfect with both hands and feet.

"Right upper limb slightly feebler than the left, especially the hand and fingers. Slight unsteadiness of right upper limb with finger nose test. Some clumsiness in buttoning his clothes with his right hand and in using a knife at meals.

"Right wrist-jerk and right knee-jerk brisker than left. Ankle-jerks equal. Plantar reflexes flexor; abdominals brisk and equal."

He first came under my care on *February* 1, 1916, *six weeks after the injury*. The wound was firmly healed and consisted of a round opening in the skull, which pulsated strongly. It extended from 14·5 to 18 cm. behind the nasion. The upper border was 4 cm. and the lower border 7 cm. to the left of the middle line. The opening was 3·5 cm. in length and 3 cm. in breadth vertically. The total distance between the nasion and inion was 34 cm.

He was highly intelligent and an excellent subject for examination. He did not suffer from headaches; speech was normal and he had had no convulsions or seizures.

Vision was perfect and the discs showed no signs of past or present changes. The pupils reacted normally, ocular movements were well performed and face and tongue were unaffected.

Reflexes.—The right wrist-jerk was brisker than the left, but otherwise the reflexes were normal and equal. The abdominals were active in all four segments and the toes went downwards, when the soles were scratched.

Motion.—There was no paralysis of any part of the hand and both grasps were strong; but his writing had changed. He used to write quickly a somewhat rounded hand; now the letters had become pointed and jerky, like those of an old man. This he thought was due to the condition of the wrist and not to any fault in the fingers. When he held out his hands in front of him, the fingers were in alignment and individual movements were perfect. There was no inco-ordination with the eyes open, but when they were closed the movements of the limb as a whole were slightly ataxic. The fingers were neither hypotonic nor spastic, and the tone of the arm was not obviously increased apart from the exaggeration of the right wrist-jerk.

His gait was normal and the lower extremity was not in any way affected.

Sensation.—He complained that the right elbow "felt odd and heavy" compared with the left. The hand seemed to him perfectly normal.

Measured movement.—I could find no loss of appreciation of passive movement anywhere except in the right elbow and wrist-joint. In all the digits and at the shoulder-joint, he responded perfectly to an excursion of 1°. But, at the right wrist-joint, it was necessary for a movement to reach 3° to 4° before it was appreciated. At the right elbow-joint he showed all those deviations from normal so characteristic of a cortical lesion; not only was the threshold raised to between 7° to 10°, but hallucinations of movement frequently disturbed the records. This never occurred on the normal side, where he appreciated a movement of 1° or less with extreme accuracy.

Vibration.—The tuning-fork showed no abnormality over the digits on the radial aspect of the wrist. But there was profound shortening of from ten to twelve seconds over the right elbow-joint. This was appreciable to me; for he called out that the fork had stopped beating on the right elbow long before it ceased to be perceptible to my fingers. This was not the case when it was placed on the left elbow. The shoulder-joint gave equal readings on the two sides.

Compass-test.—The fingers and palm yielded equally good records on the two sides, when the points were separated to 1 cm. Over the normal (left) forearm he did not give a perfect series of answers until they were 4 cm. apart;

3 cm. was distinctly below the maximum threshold. But on the right forearm he showed complete want of discrimination and confusion between one and two points even at 10 cm. The two soles gave a perfect series of answers at 3 cm.

Localization was not affected on the hands; but he seemed to be more uncertain in the neighbourhood of the right than of the left elbow. This proved to be the case at a subsequent more detailed examination.

Tactile hairs.—Over the fingers and the palm of the hand he reacted perfectly to a hair of 21 grm./mm.^{2}, but tactile sensibility was obviously affected over the flexor aspect of the forearm. On the normal side the response to 21 grm./mm.^{2} was not quite certain and it required a hair of 23 grm./mm.^{2} before he gave a perfect series of answers. But, over a similar part of the right forearm, even 35 grm./mm.^{2} failed to evoke a sensation with every contact. He said "the left is easier and I can be more certain on the left arm."

The soles gave perfect records with 21 grm./mm.^{2}

Thermal-tests.—The hand was unaffected, and I could find no certain change of sensibility over the forearm in the neighbourhood of the flexure of the elbow. But he said, "It takes some time over the right elbow before I recognize that there is anything there at all. When I have recognized it, I begin to feel the temperature."

Prick.—I could find no difference in sensibility to prick between the two upper extremities.

Weight.—There was no difference between the two hands in the power of discriminating weights.

This patient was examined on several subsequent occasions and all the observations pointed to affection at the right elbow without disturbance of the hand or shoulders. I shall pass on, however, to his condition in *June*, 1918, *two years and six months after the injury.*

He was in excellent physical condition and had had no convulsions or seizure. From time to time, about once a month, he suffered from headache, always connected with some increase of pulsation in the scar. Running upstairs or excessive heat would bring on the headache, which passed off when he lay down.

The scar was firm, tough and somewhat depressed; it did not pulsate as he sat upright in a chair.

Reflexes.—All the reflexes were normal and equal except the elbow- and wrist-jerk, which were distinctly greater on the right than on the left side.

Motion.—The grasps were equal, but his handwriting still showed the same changes, though to a less degree than before. He used to be a good lawn tennis player, but was now "terribly bad"; "I hit the ball on the wood and sometimes miss it altogether." Golf has not suffered nearly so much.

He is a strongly right-handed man and, although there is no obvious paresis of the right arm against resistance, the force exerted is not greater than that of the left, when bending or straightening the elbow. He is using his left arm more and more because he finds the right arm clumsy. When he held his arms out in front of him, the fingers were in perfect alignment and did not fall away on closing his eyes; but the arm swayed somewhat and was less steady than the left. He could touch his nose perfectly with the right forefinger when his eyes were open; as soon, however, as they were closed, he became uncertain in aim and struck some part of his cheek or hit his nose too hard. There was no obvious change in tone.

Sensation.—He complained that "the feeling in the right elbow is very peculiar; I do not feel when people knock up against me in a crowd as I do with the left arm. I get a sensation of some sort, but it is not a normal sensation. It would be more accurate to say that someone could bump up against me, and I could not tell what it was that touched me."

Measured movement.—The index and little fingers, wrist and shoulder responded perfectly to 1° of passive movement. The right elbow required from 1·5° to 3°, and he said, "It feels quite different in this elbow,"

Compass-test.—Perfect readings were obtained with a distance of 1 cm. over the index and little fingers and palm of the right hand. Over the right forearm the threshold was 6 cm., whilst over a similar part on the left side he gave a perfect series of answers at 4 cm.

Localization was equally good over the two hands, but was affected on the flexor surface of the right forearm in the neighbourhood of the elbow-joint. A series of observations were made in which he indicated the spot touched on the arm of an assistant. Out of ten contacts he made two distinct errors on the normal (left) forearm, whilst on the affected limb one only of the stimuli over approximately similar spots, was accurately localized. Three contacts in the neighbourhood of the right wrist were indicated correctly.

Tactile hairs.—The fingers and palm of the right hand gave perfect readings with 21 grm./mm.^2; this hair was also appreciated over the flexor aspect of the normal elbow. But over a similar part of the right arm 35 grm./mm.^2 was necessary before he responded to every contact.

The difference between the two limbs was evident, when a record was taken with a hair of 21 grm./mm.^2 in the following manner: First a few contacts were made on the normal, and then a similar number on the affected arm, changing from side to side until sixteen stimuli had been applied to each in turn. The following results were obtained:—

Left elbow ..	l.l.l.l.		l.l.l.l.l.l.		l.l.l.l.l.l.	
Right elbow ..		o.l.l.l.		o.l.o.o.l.l.		l.l.o.o.l.o.

At the end of these observations he said: "Here again the principal difference is that I am not so certain on the right arm."

Weight, form and *texture* were all perfectly appreciated on the right hand.

Case 15.—*Wounded August* 20, 1916, *over the vertex in the parietal region. The wound extended slightly more to the right than to the left of the middle line. This case is remarkable because the three aspects of cortical sensibility were dissociated; spacial recognition was disturbed in the left foot and ankle, tactile and thermal discrimination were defective in the right foot, whilst the power of appreciating similarity and difference was not affected on either side. The reflexes were normal and individual movements were carried out equally well in both feet. The left great toe was definitely hypotonic.*

Lieutenant E. D. J., aged 22.

He was wounded on August 20, 1916, with a piece of shell-casing which perforated his helmet. He fell and became unconscious for a few seconds. Two hours later he vomited. He had very little headache at the time. From the first the left leg "felt odd"; he wanted to step over a man in the trench and stepped on to him. No operation was performed at any of the hospitals through which he passed before he came under my care.

He was admitted to the London Hospital on September 2, 1916, without a note of any kind.

The wound consisted of a linear cut across the middle line of the scalp, with a suppurating sinus leading down to an extensive area of bare bone. The anterior border of this opening lay 21 cm., and the posterior border 22 cm., from the root of the nose. It extended 1 cm. to the right and 2 cm. to the left of the middle line. The total nasion-inion measurement was 34 cm. A radiograph showed flaking of the inner table of the skull and a small fragment of metal superficial to the bone.

The following observations were made between *September* 2 *and* 20, 1916.

He was an intelligent man, who had been an accountant in civil life He suffered from little or no headache, and had had no convulsions or seizures of any kind. Speech was normal.

He was slightly myopic, but on correction his vision was $\frac{6}{6}$. The visual fields were normal.

Pupils, movements of the eyes and those of the face and tongue were unaffected.

All the *reflexes* were normal, both superficial and deep.

Motion.—The upper extremities were entirely unaffected.

He could walk well and his gait was not obviously changed, though he complained that the left foot was still clumsy; for instance, he found it impossible to put on his slippers if he was standing up.

There was no paralysis, and all movements of the toes could be carried out as well on the one foot as on the other.

So long as his eyes were open, he could stand well on either foot and there was no obvious inco-ordination. But on closing his eyes he was distinctly more unsteady on the left than on the right foot and movements of this leg were somewhat ataxic.

The most striking difference between the two feet was the hypotonic condition of the left great toe. Thus, when pushed up, it could be extended passively so as to subtend less than a right angle with the line of the dorsum of the foot. The mobility of the right great toe was noticeably less; it could be pushed up to form an angle of about 120° only.

There was no obvious wasting in either lower extremity.

Sensation.—The two feet seemed to him quite different. The left was "clumsy" and there was a "fat feeling under the toes." But this foot was "quicker in picking up a cold feeling" than the right.

The two hands were normal to all the tests for sensation.

Measured movement gave the following results:—

	Right	*Left*
Great toe	1° to 2°	3° to 5°
Ankle .. .	1°	2° to 3°
Knee	1°	1°

Vibration.—When the tuning-fork was placed over the ball of the great toe, there was a shortening of from 7 to 10 seconds against the left foot; but over the inner malleolus there was no definite difference. This supports the results of examination by means of passive movement.

Compass-test.—The records obtained with this test were of great interest; for they showed the difference in the records due to want of two-dimensional discrimination and to unequal tactile appreciation. I give here the actual readings, where a stroke represents a correct and a cross a wrong answer. These have been translated into numbers on p. 128.

Right sole (tactile loss)

3 cm.	1	ll lxl lxlxx
	2	l lxxl lxlxx
4 cm.	1	xxxl lxll ll
	2	lxxx lxl lxl
6 cm.	1	lxxl llllxl
	2	xlxx llxxxl
10 cm.	1	ll lll l ll ll
	2	lll l l lll ll

Left sole (spacial loss)

3 cm.	1	llll llll l
	2	lxxl llxxl l
4 cm.	1	lll lll llll
	2	ll ll lxll ll

Localization was unusually good on both feet and I could discover no difference between them; but it must be remembered that our method of testing localization, applied to the feet, can reveal coarse changes only.

Tactile hairs.—The contrast between the condition of the two soles to this test was striking.

	Right sole (affected)	*Left sole* (unaffected)
21 grm./mm.^{2}	looloooollooooo	
28 grm./mm.^{2}	olololllllooooo : : —ol : :	
35 grm./mm.^{2}	llollolooooololll	
70 grm./mm.^{2}	lolollolooloooolo	
100 grm./mm.^{2}	oloooollolololll	
21 grm./mm.^{2}	loooolooloololol	
21 grm./mm.^{2}	—	llollollllololll
29 grm./mm.^{2}	—	lllllllllllllll

He complained that on the right sole he had great difficulty in knowing when the hair was put on or taken off; "It seemed like a feather rubbed over the foot." On the left sole the sensation was normal.

Thermal test.—I could not find any definite difference between the threshholds for heat and cold, but at this time I carried out no comparative observations on the sole [*vide infra* p. 228]. The determination of thermal thresholds on the foot is extremely difficult, and often yields unsatisfactory results unless the greatest precautions are taken to prevent exposure. In some patients the feet are naturally cold, even in bed.

Prick.—The threshold for prick was the same on two soles (3 grm.).

Form.—He could recognize all these tests when he was allowed to roll them under his feet against the ground.

On September 27, 1916, Mr. Walton trephined over the area of the wound and removed an irregular piece of the inner table of the skull; it lay loose on the dura, which was not perforated. The longitudinal sinus was directly to the right of the situation in which lay the loose fragment. Some pieces of metal were removed from the neighbourhood of the outer table. The wound finally healed on November 18, 1916.

I was able on several occasions to confirm the existence of the remarkable dissociation of sensibility given in detail above; but it will be sufficient to add a short account of observations made during the *last week of February*, 1917, *six months after the injury.*

His general condition was excellent. His memory and attention were good, and he was able to read with interest, and remember what he had read. He was entirely free from headache, and had had no convulsions or seizures of any kind.

The *reflexes* were in every way normal and equal.

Motion.—Movements of the toes were as well executed on the one side as on the other, and the only abnormal condition was some hypotonia of the left great toe. This was, however, considerably less than at the first examination. He could walk seven miles, but after about five miles noticed that his left foot became tired and dragged.

Sensation.—He still had "a curious abnormal feeling" in the left foot, especially "over the toes and pads, where the front of the foot touches the ground."

Measured movement.—He answered quickly and accurately to passive movement of 1° in the right great toe; on the left side he required a range of 2°, and was slow in his replies. The left ankle was now normal, and he responded to movements of 1°.

Compass-test.—On the right sole he still complained that "a touch seemed to linger, and it is difficult to find out if it is the one you put on last or the two points together." He gave an almost accurate reading, however, at 6 cm.

On the left sole he responded with a perfect series at 3 cm., the smallest distance that can safely be applied in clinical examination.

Localization was equally good on the two feet; he made no mistakes on either side.

The *tactile hairs* revealed exactly the same changes as before. He complained: "I seem to be longer in feeling it on the right foot. My difficulty was the uncertainty of contact."

On the right sole a good reading was obtained with 70 grm./mm.^{2}, marred, however, by a tendency to hallucinations; all hairs of lower bending strain showed obvious defects of sensibility. On the left sole the condition was exactly as before; 23 grm/mm.^{2} was perfectly appreciated, whilst with 21 grm./mm.^{2} he did not recognize all the sixteen stimuli. He was certain that, as far as this test was concerned, the left foot was normal.

Thermal tests.—I devoted one sitting to a series of observations on thermal sensibility. I could find no definite change in the thresholds for heat and cold, but the foot is so unsatisfactory a situation for tests of this kind, and his answers were so much slower from the right than from the left sole, that I carried out a series of comparative observations. He had no difficulty in recognizing the relative warmth of two silver tubes at 35° and 44° C. on the left sole; but on the right, out of seven observations, he was wrong four times, once doubtful and twice correct in his answers. Not only was he slower, but said, "I feel uncertain in the right foot; it takes me longer to grip it."

Prick.—I could find no difference in the threshold on the two soles.

Weight.—On a previous occasion a careful series of observations had been made by adding and subtracting weights, placed on the dorsal aspect of the basal phalanx of the great toe; the soles rested flat on the bed and were fully supported. He was remarkably accurate in his replies, when the test was carried out in this manner, and I could find no difference between the aptitude of the two feet.

Form.—He had no difficulty in recognizing the various test-objects when he was allowed to roll them under either foot. He could discover no difference in the sensations from the two soles.

Case 18.—*This case is a remarkable example of dissociated sensibility due to a lesion of the cortex. The tactile hairs showed distinct sensory loss over the thumb, index and middle fingers, although the power of appreciating passive movement and vibration was perfectly preserved. The compass-test was defective in consequence of the loss of tactile sensibility. Individual movements of the three pre-axial digits were clumsy. There was no hypotonia.*

Colonel M., aged 46.

On October 4, 1914, he was hit in the right parietal region by a rifle bullet. It must have been a glancing blow, for the bullet was found beside him.

He was admitted to the Allied Forces' Base Hospital, and Mr. Whitehall Cole kindly furnished the following report:—

"The patient was fully conscious on admission, but showed definite signs of cerebral irritability. He was hemiplegic, chiefly in the left arm and face; the leg was slightly affected only.

"At the operation on October 23, definite cerebral substance could be seen to protrude through the scalp. Several small fragments of bone lay loose in the wound. The dura showed a small lacerated wound from which there was slight exudation of cerebral substance. All loose fragments of bone were removed and the osseous opening was slightly enlarged sufficiently to ascertain that no depressed bone remained. The wound was closed except for a gauze drain passed down to the dura.

"Recovery was steady and uneventful. The leg cleared up rapidly. The face began to improve after the operation and progressed steadily. The arm made very little progress until the last week or ten days of his stay, when his grip became stronger. There was no return of movement at the elbow and wrist."

The leg recovered completely and his general condition became normal; but the thumb and index finger of the left hand showed no improvement.

On June 23, 1915, he had an epileptiform attack associated with an abnormal "feeling" in the left arm, followed by a short period of unconsciousness.

On June 28, 1915, he was admitted to the Palace Green Hospital for Officers under my care.

The following observations were made *between July* 14 *and July* 20, 1915, 283 *to* 289 *days after the injury.*

The wound was perfectly healed; it was represented by a small opening in the skull 2 cm. horizontally and 1·5 cm. vertically in the right temporal region. The anterior border of this trephined area corresponded with a point 15 cm. back from the nasion and 10 cm. to the right of the middle line. The total nasion-inion line was 35·5 cm. in length. The opening was depressed and did not pulsate. A radiograph showed no abnormality beyond the opening in the bone.

He was an unusually intelligent man of intellectual tastes and an excellent subject for examination.

Speech was normal. He suffered from no attacks or seizures beyond the one already described. He was free from headache, nausea or vomiting.

Pupils and ocular movements were normal.

There was slight weakness of the lower portion of the left half of the face and the tongue tended to turn to the left on protrusion.

Reflexes.—The left arm-jerks were brisker than those from the right upper extremity. The left knee-jerk was slightly more active than the right. The plantar reflexes were normal, but the response from the left lower segment of the abdomen was undoubtedly sluggish compared with that from the right.

Motion.—When he held both arms in front of him, the digits of the left hand were not in alignment. On attempting to abduct them they fell into great disorder. Isolated and individual movements of the fingers and thumb were badly executed; they were slow, clumsy, and ineffective.

All movements of the wrist and elbow were strongly performed against resistance.

When the hands were held out and the eyes were closed, the left wrist became slowly flexed and the fingers fell out of place. There was definite ataxy, when he attempted to bring his left forefinger to his nose

No hypotonia and no tonic rigidity could be discovered in any part of the left upper extremity.

There was no wasting of the hand or arm.

The left lower extremity was in every way normal.

Sensation.—

	Left (affected) hand					*Right hand*
	Thumb	Index finger	Middle finger	Ring finger	Little finger	—
Measured movement	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°
Vibration	Perfect	Perfect	Perfect	Perfect	Perfect	—
Compass-test	Affected at 3 cm.	Affected at 3 cm.	Affected at 3 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.
Localization	Perfect	Perfect	Perfect	Perfect	Perfect	Perfect
Tactile hairs	Threshold with 35 grm./mm.^{2}	Gross loss with 100 grm./mm.^{2}	Threshold with 23 grm./mm.^{2}	Perfect with 21 grm./mm.^{2}	Perfect with 21 grm./mm.^{2}	Perfect with 21 grm./mm.^{2}
Thermal ..	Not tested	Defective	Not tested	Not tested	Not tested	Perfect appreciation of 35° and 40° C.
Prick ..	Not affected	Not affected	Not measured	Not measured	Not measured	—
Weight ..	Not tested	Gross want of appreciation over these two fingers together		Perfect appreciation over these two fingers together		—

The left lower extremity was in every way normal.

These observations are remarkable for the perfect readings given by the test for passive movement and vibration in spite of the obvious affection of tactile sensibility. The compass-test was disturbed from the latter cause (p. 128).

I had the opportunity of examining him carefully on several subsequent occasions, but shall only append a short summary of observations made on *September* 26 *to* 28, 1915, 357 *days after the injury.*

He had had no further attacks, and had suffered from no headaches.

There was still slight weakness of the left angle of the mouth, and the tongue tended to deviate to the left on protrusion.

Reflexes.—The left wrist-jerk was distinctly brisker than the right, but the knee-jerks and ankle-jerks were normal. The plantar response was normal, and the left lower segment of the abdomen responded briskly to stimulation.

Motion.—He complained that he could not use his fork properly, and could not use his left hand to do up his boot-laces. "I have no grip between the thumb and first finger, and have to hold my fork between the middle and ring fingers."

He could abduct, extend and flex the thumb, but the movements were clumsy and were nearly always associated with motion in some other digit. The same want of co-ordinate sequence was seen in movements of the index and middle, but not in those of the ring and little fingers.

There is no increase or decrease of tone and no wasting.

Sensation.—

	Left (affected) hand					*Right hand*
	Thumb	Index finger	Middle finger	Ring finger	Little finger	—
Measured movement	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°	Perfect at 1°
Vibration	Perfect	Perfect	Perfect	Perfect	Perfect	—
Compass-test	Affected at 3 cm.	Affected at 3 cm.	Not quite perfect at 3 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.
Localization	Perfect	Perfect	Perfect	Perfect	Perfect	—
Tactile hairs	Threshold with 23 grm./mm.^{2}	Threshold with 23 grm./mm.^{2}	Slow with 21 grm./mm^2	Perfect 21 grm./mm.^{2}	Perfect 21 grm./mm.^{2}	Perfect 21 grm./mm.^{2}
Prick ..	Not affected	Not affected	Not affected	Not affected	Not affected	—
Weight ..	Not tested	Gross want of appreciation over these two fingers tested together		Perfect appreciation over these two fingers together		—

Case 19.—*This is an example of the third form of sensory dissociation, which may occur in consequence of a cortical lesion. The power of recognizing similarity and difference of weight and form was not disturbed in spite of considerable loss of the other two sensory groups. Wounded December* 6, 1917, *with a machine-gun bullet which produced a gutter fracture in the left parietal region. Trephined four hours later, when the dura was found to be intact but bruised and blue. It was incised and blood-clot and brain-pulp evacuated. Isolated movements of the right hand perfect with his eyes open. Distinct hypotonia of little and ring fingers. Reflexes unaffected.*

Second Lieutenant D. P., aged 23.

On December 6, 1917, he was shot down from the air, when patrolling over the lines. He must have been unconscious for about two minutes, but when he came to himself his machine was flying normally; on trying to control it he found his right hand was useless. He saw his hand upon the lever, but thought it belonged to his observer: he then appreciated that it was his own, and replaced it by his left hand. The foot and leg was not affected in any way. He brought the machine down safely and walked to the dressing station. His head was bleeding very little, he did not vomit, and suffered from no headache.

He arrived at the Casualty Clearing Station about four hours later, and was operated upon at once. The dura was perforated; blood-clot and brain matter were evacuated. Wound closed.

This was followed by much headache and some vomiting. Speech was not affected.

On December 12, 1917, the incision was opened up and some blood-clot washed away. The dura was pulsating and was not tense. The wound was left open for drainage.

On February 23, 1918, he was admitted to the R.F.C. Hospital, Bryanstone Square, and came under my care. At that time the wound had healed except for a minute spot which required dressing for three days only.

The wound was represented by a longitudinal incision on the left side of the head extending from the parietal eminence forwards almost into the frontal region of the scalp. At the posterior end of this line was a gutter in the bone. This began posteriorly 21·5 cm. from the nasion; in front it ceased 17·5 cm. from that point. It lay from 4 to 4·5 cm. to the left of the middle line. His head was unusually long, and the total nasion-inion line measured 36 cm. The scar was not pulsating and the total extent of loss of bone measured not more than 4 cm. longitudinally and from 1 to 1·5 transversely.

The following observations were made *between March* 10 *and* 24, 1918, *from* 94 *to* 108 *days after the injury.*

His mental condition was excellent and he was an accurate and most willing observer.

He was entirely free from headache, and had not suffered from convulsive attacks or seizures of any kind.

The pupils were normal. Movements of the eyes, face and tongue were perfect.

All the *reflexes* of both upper and lower extremities were normal, and equal on the two sides.

Motion.—The grasp of the right hand was distinctly weaker than that of the left, but all individual movements of the fingers could be performed perfectly with the eyes open; when they were closed the tip of the right thumb was not approximated to that of middle, ring or little finger with the same precision as in the left hand.

All movements of the right wrist, elbow and shoulder were extremely powerful.

If the hands were held steadily in front of him there was no want of alignment of the fingers, even when his eyes were closed.

There was distinct hypotonia of the little and ring fingers of the right hand. The other digits seemed to be normal. There was no wasting. The right lower extremity was in every way normal.

Sensation.—He said, "I can use the thumb and first finger; the trouble is mostly in the three other fingers. It is a slight stiffness or numbness, as if I had a tight glove on." He could not play the piano with the right hand: "The fingers won't get on to the right notes." He was able to button his clothes, but found it somewhat difficult with the right hand.

	Right (affected) hand					*Left hand*
	Little finger	Ring finger	Middle finger	Index finger	Thumb	—
Measured movement	15° to 20°	10° to 15°	4° to 8°	2° to 3°	1° to 2°	Perfect at 1°
Vibration	8 sec.	8 sec.	8 sec.	5 sec.	2 sec.	—
Compass-test	Gross loss at 3 cm.	Threshold, 3 cm.	Introspective, 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.	Perfect at 1 cm.
Localisation	One error; very slow	Slow	Normal	Normal	Normal	No errors
Tactile hairs	28 grm./mm.^{2}	23 grm./mm.^{2}	21 grm./mm.^{2}	14 grm./mm.^{2}	14 grm./mm.^{2}	Perfect with 14 grm./mm.^{2}
Thermal ..	Gross loss	Slow; one error	Normal	Normal	Normal	Perfect appreciation of 35° and 43° C.
Prick ..	10 grm.	9 to 10 grm.	8 grm.	8 grm.	8 grm.	8 grm.
Weight ..	Perfect	Perfect	Perfect	Perfect	Perfect	Perfect series
Form ..	No loss with these two together		—	No loss with these two together		—
Texture ..	Affected	Affected	Introspective	Perfect	Perfect	Perfect recognition

The right lower extremity was in every way normal.

Measured movements.—All five digits of the right hand were affected; the loss of sensibility was extremely slight in the thumb, but increased until it reached considerable proportions in the little finger.

Vibration.—This test corroborated the results obtained by passive movement.

Compass-test.—There was gross loss over the little finger, when the points were 3 cm. apart; but over the ring finger a threshold could be obtained at this distance. The change in the middle finger was introspective only; he found it harder to make up his mind, although his replies were correct. The index and thumb were not affected.

Localization.—He made one definite error only, which consisted in localizing a touch over the basal phalanx of the little finger on the same phalanx of the ring finger. But in this case the slowness of answering over the little and ring fingers compared with his quickness over the other digits gave a better idea of the disturbance of localization than the records. He himself said, "I have difficulty in making up my mind; when you touch me on the ring finger I cannot be certain whether you are touching me on the ring or middle finger." But he finally succeeded in choosing the correct phalanx and the right digit, except once on the little finger.

Tactile hairs.—With a 14 grm./mm.^{2} hair he said there was no difference between the sensation on the thumb and index fingers of the two hands. On the middle finger the threshold was raised to 21 grm./mm.^{2}, and he said it was "clearer" on the left than on the right hand. When a hair of 21 grm./mm.^{2} was applied in the usual way to the ring finger he not only failed to recognize the contact eight times out of sixteen, but said, "It doesn't seem like a sharp point at all; it is as if you were touching me with your finger." The condition was similar over the little finger of the right hand.

Thermal.—When two silver tubes containing water at 20° C., and at 40 °C., were applied to the little and ring fingers of the two hands in varying order, he did not fail to name them correctly, but said that 20° C. was "colder" and 40° C. "warmer" on the normal than on the abnormal digits.

Two tubes containing water at 35° and 43° C. were correctly recognized over the thumb and index finger of the right hand. Over the middle finger his replies were also accurate, but he complained that it was "much easier" on the normal than on the affected hand. Over the right ring finger his answers were slow, and he made one mistake in four observations. Over the right little finger he could detect no difference between the two tubes, but named them correctly on the little finger of the normal hand.

The threshold was raised on the right little finger both for heat and for cold. For cold it was about 25° C. on the right and 27° C. on the left hand, whilst for heat it was about 36° C. on the right, and 29° to 30° C. on the normal side. Thus, the changes consisted in a considerable enlargement of the neutral zone. A similar condition, to a less degree, was present over the right ring finger; but on the middle fingers the thresholds were identical.

Prick.—Unfortunately, the threshold over the tips of the normal fingers was rather high to this test, and he did not recognize the painful element in the prick under about 8 grm. on our algesimeter. But, although there was no difference between the thumb, index and middle fingers of the two hands some loss of sensibility was present in the ring and little fingers on the right side. This came out particularly well when the instrument was set at 8 grm. and a series of pricks were made over the right and left little and ring fingers; they were said to "prick" on the left, but to be "blunt" on the right hand.

Weight and Form.—The striking feature of this case was his perfect sensibility to these tests. He was not conscious of any difference between the two hands, even over the little and ring fingers.

Texture.—Over the thumb and index finger there was no difference between the two hands. Over the right middle finger the four textile objects were named correctly, but he complained that he had some difficulty in being certain, "because they were not so sharp" on the right hand. Over the ring finger he made several mistakes, and said, "I can tell it's a bit of cloth, but I can't feel if it is rough or smooth so well on the right hand." On the right little finger he said it was "very difficult to tell what kind of thing it was," but named two out of the four stuffs correctly. Evidently this is one of the cases where the accuracy of this test suffers through defects of tactile sensibility.

Case 23.—*An example of a combined cortical and subcortical injury. Wounded November 14, 1917, by a rifle bullet in the right parietal region. Cerebral abscess evacuated on December 8. The wound had healed before he was admitted to the London Hospital on February 6, 1918. The main feature of the case was the hypotonic condition of the left hand, associated with a peculiar change in its contours and general appearance. The maximum loss of sensation lay over the index and middle fingers.*

Private W., aged 22.

On November 14, 1917, he was hit by a rifle bullet, which penetrated his helmet. He dropped and became unconscious for a few minutes, but was then able to walk about 200 yards to a dugout. He reached the Base Hospital about nine hours later, and the wound was excised and sutured at once. Vision and speech were not affected, and he suffered little from headache during the first twenty-four hours. But the headache then became very severe, and by December 4 he had begun to vomit. Four days later the note on the field card said: "Going wrong. Cerebral abscess. Headache and vomiting. Weakness of left face and arm. Slow pulse; subnormal temperature." On December 8 he was operated upon, and a very small fissured fracture exposed. When the bone was removed, pus welled up through a hole in the dura mater. A small tube was inserted, and the flow of pus became profuse. From this time his progress towards recovery was uninterrupted.

He was admitted to the London Hospital on *February* 6, 1918, *eighty-four days after the injury* and sixty-four days after evacuation of the cerebral abscess.

All the wounds were healed, and the trephined area was somewhat depressed. The site of the original injury was represented by an almost horizontal scar, 8 cm. in length, in the right parietal region [fig. 1]. This was bisected by a short transverse cut, apparently the result of the first operation. The central portion of the wound, which lay directly over the trephined area, was 18·5 cm. along the nasion-inion line and 9 cm. to the right. His head was remarkably big, measuring 37 cm. from the root of the nose to the level of the the external occipital protuberance. The trephined area was 5 cm. in length and 4 cm. in breadth, and of an oval shape; measured along the nasion-inion line, the anterior border of the opening was 17 cm. and the posterior 22 cm. It pulsated heavily.

His mental state was remarkably good, speech was unaffected, and he had little or no headache so long as he was kept in bed. The headache became somewhat severe after he was allowed to get up, and he would suffer from a severe attack every two or three days. There was no nausea or vomiting, and he did not suffer from convulsions or seizures.

The discs, pupils, and ocular movements were in every way normal. Movements of the face, tongue, and palate were unaffected.

All the reflexes were equal and normal on the two sides.

Motion.—The grasp of the left hand was somewhat weaker than that of the right in the relation of 30 to 50, but all individual movements could be carried out by each digit in turn so long as the eyes were open. When they were closed, however, individual movements became clumsy: he succeeded in approximating the thumb to each finger by holding the fingers together in a flexed position and bringing the thumb into contact with the tip of each in turn. Moreover, the thumb slid off one finger to assume its position on the next, and the rhythm of the movement was defective. When the hands were held out in front of him, the fingers were in alignment, but, on closing the eyes, the little finger drooped; the other digits were abducted and adducted irregularly, and were no longer under perfect control. He could bring his

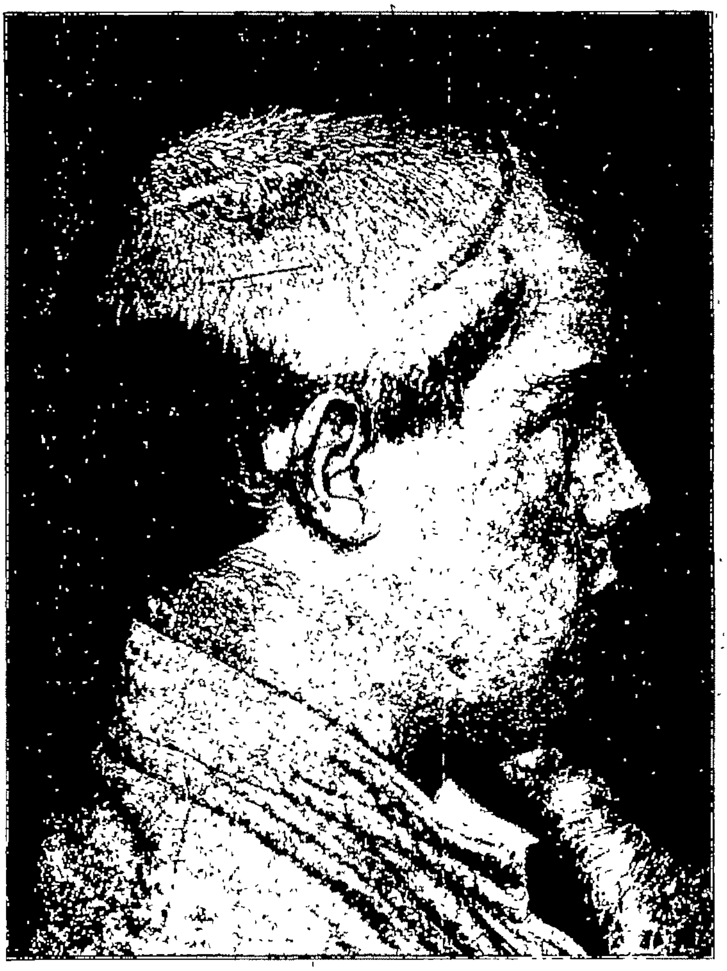

FIG. 1.

finger to his nose perfectly with his eyes open; but when they were closed it was liable to strike the cheek, or to arrive clumsily on the spot at which he was aiming.

There was no gross wasting, but the difference between the two hands was profound [fig. 2]. The left hand resembled that of a woman, and was soft and rounded with delicate fingers; the right was that of a man capable of hard manual work. Its surfaces were squarely modelled, the palm broad and flat and the fingers had an appearance of shortness in relation to the hand, as a whole. The left hand, on the contrary, was rounded and soft; its lines were less deep. The fingers seemed to be longer and thinner than those on the normal side, although by measurement they were actually a little shorter. This illusory appearance was due to the smaller width of the left palm, which measured 8 cm. compared with 9 cm. on the right hand. The tips of the fingers were spatulate on both hands, but less so on the left than on the right. The interosseous spaces were not wasted; they seemed, however, on the affected side to be occupied by smaller and softer muscles. The patient had done no manual work for nearly four months, when the photographs were taken, and both hands were white and free from corns. The difference between them was immediately evident on contact. In the act of shaking hands, the

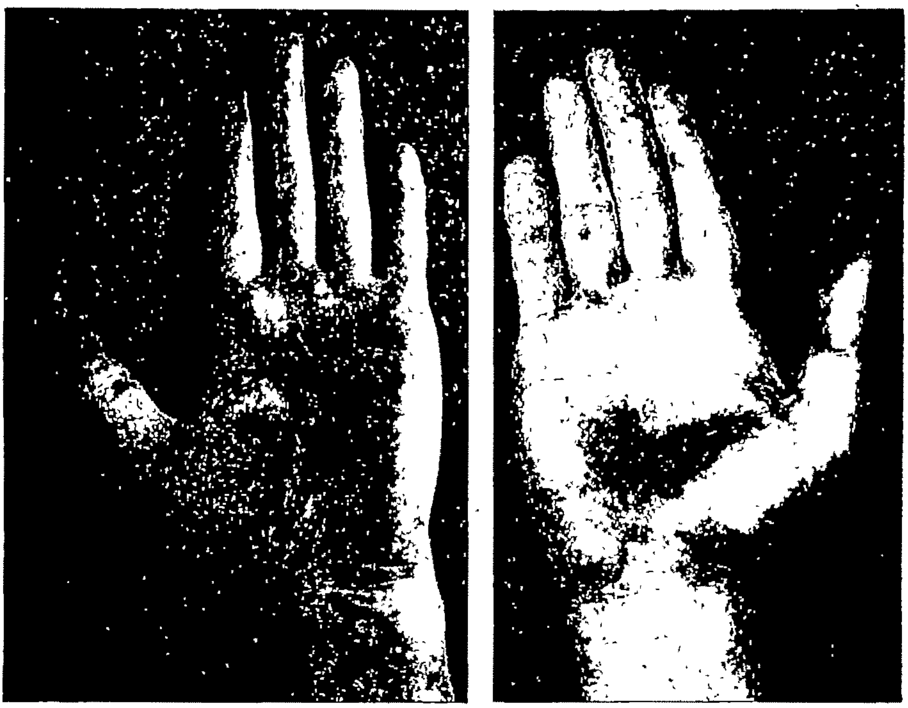

FIG. 2.—To show the condition of the right and left hands in No. 23.

right grip was firm and well sustained. I was conscious of the elastic pressure of the contracting muscles of the palm and of the tension of the fingers; I had the feeling that if the patient chose to increase his grasp it might become painful. But when I gave my left hand into his, it was like the impression received from the hand of a woman. My fingers seemed to be in contact with something soft, and without resistance; the pressure was orderly, but gentle and harmless.

All the digits of the left hand could be bent back passively one by one to an abnormal extent; even the thumb seemed to be hypotonic.

There was no paralysis of the lower extremities, and movements of the left hip and knee could be carried out strongly against resistance; but dorsiflexion, and to a less degree plantar extension, of the left foot were not so forcible as on the right side. All movements of the toes were possible. He could walk well and could stand on the right foot, both with his eyes open and shut; on the left foot he could stand unsteadily but fell when his eyes were closed. I could find no definite signs of hypotonia in the left foot. There was no spasticity.

Sensation (between *February* 11 *and March* 10, 1918, *eighty-nine to* 116 *days after the injury*).

He complained that the left hand "felt numb," and this abnormal sensation was worse over the palm and forefingers than over the thumb and thenar eminence. When he woke at night it seemed to him as if he had lost the index and middle fingers; "this part of the hand seemed gone altogether." He did not recognize any abnormality in the left lower extremity.

The results obtained were the usual sensory tests at this time as shown in the following table:—

	Left (affected) hand					*Right hand*
	Thumb	Index finger	Middle finger	Ring finger	Little finger	Normal digits
Measured movement	10° to 20°	10° to 20°	20° to 30°	15° to 25°	20° to 30°	1°
Vibration	Affected	Affected	Affected	Affected	Affected	—
Compass-test	Gross loss at 3 cm.	Gross loss at 3 cm.	Gross loss at 3 cm.	Gross loss at 3 cm.	Gross loss at 3 cm.	Perfect at 1 cm.
Localization	Not affected	Affected	Affected	Not affected	Not affected	No mistakes
Tactile hairs	23 grm./mm.^{2}	Not perfect 100 grm./mm.^{2}	No threshold 100 grm./mm.^{2}	Not perfect 70 grm./mm.^{2}	Not perfect 70 grm./mm.^{2}	Perfect 21 grm./mm.^{2}
Thermal ..	Affected	Affected	Affected	Affected	Affected	Perfect recognition of 35° and 40° C.
Prick ..	5 grm.	Gross affection	Gross affection	8 grm.	8 grm.	4 grm. threshold
Weight ..	Affected	Gross loss	Gross loss	Considerable loss	Considerable loss	Perfect appreciation of the series of changes in weight
Texture ..	Affected	Affected	Affected	Affected	Affected	Perfect recognition

He was sent to a convalescent home, and, on his return, the following observations were made between *May* 6 *and* 12, 1918 (173 *to* 179 *days after the injury*).

His headache had almost entirely ceased; he had given up smoking, and, with the disappearance of the chronic cough, the most potent cause of his headache was removed.

Motion.—Individual movements of the left hand could be carried out so long as his eyes were open, but, when they were closed, the fingers became somewhat ataxic, especially the index and middle. These two digits were still hypotonic, but the thumb, ring and middle fingers could also be bent back to a somewhat greater extent than similar parts of the right hand.

Sensation.—The condition had improved considerably, but the index and middle fingers still showed the greatest loss of sensation.

	Left (affected) hand					*Right hand*
	Thumb	Index finger	Middle finger	Ring finger	Little finger	Normal digits
Measured movement	1°.	5°	5°	1°	1°	Perfect at 1°
Vibration	No difference	6 sec. shortening	5 sec. shortening	No difference	No difference	—
Compass-test	1 cm.	5 cm.	Lost at 5 cm.	1 cm.	1 cm.	1 cm.
Localization	Perfect	Affected	Affected	Perfect	Perfect	Perfect
Tactile hairs	Normal 21gr./mm.^{2}	Not perfect at 100 grm./mm.^{2}	Not perfect at 100 grm./mm.^{2}	35 grm./mm.^{2}	23 grm./mm.^{2}	21 grm./mm.^{2} perfect
Thermal ..	Not affected	Gross loss	Gross loss	Not affected	Not affected	Perfect recognition of 35° and 40° C.
Prick ..	4 grm.	8 grm.	8 grm.	4 grm.	4 grm.	4 grm. threshold
Weight ..	Perfect	Affected	Affected	Perfect	Perfect	Perfect series
Form ..	Perfect	Slow and uncertain	Slow and uncertain	Perfect	Perfect	Perfect recognition
Texture ..	Perfect	Gross loss	Gross loss	Introspective	Introspective	Perfect recognition

(B) APPROXIMATE POSITION OF THE LESION IN EACH CASE CITED IN THIS PAPER. (For use with Chapter VIII.)

It is impossible to determine with accuracy the position of the injury to the brain from the situation of the wound on the surface. But certain rough conclusions can be drawn, and Professor Elliot Smith has kindly attempted to localize the main incidence of the lesion in each of my cases. My sincerest thanks are due to him for the trouble he has taken and the encouragement he has given me in this matter.

Case 1.—Lieutenant A. A., aged 26. Wounded October 8, 1915, by a fragment of a high-explosive shell. He was not wearing a helmet.

He was admitted to the Empire Hospital for Officers on December 22, 1915, with the following note: "Operation on October 9, 1915, by Major Austen. Depressed bone removed. A large fracture passed forwards towards the right eye. Dura mater opened widely and 2 to 3 oz. of subdural blood and brain matter escaped."

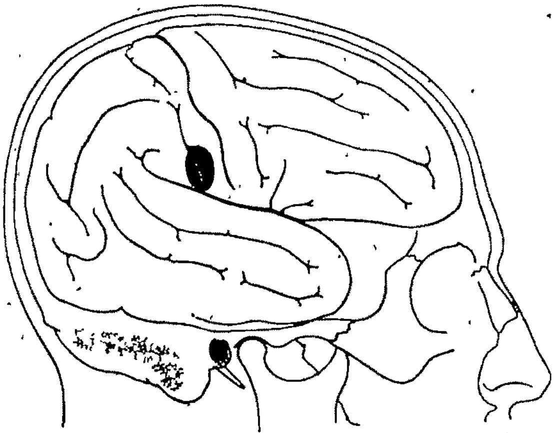

FIG. 3 (Case No. 1).

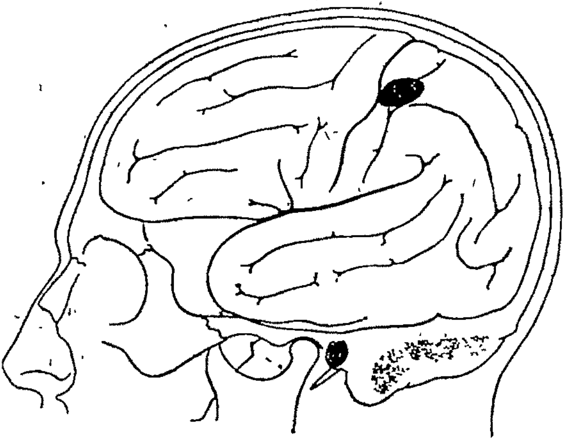

FIG. 4 (Case 2).

"The brain at first formed a slight hernia, about the size of half-a-crown; but, after about fourteen days, it sank away and left a hollow below the level of the bone."

The opening in the skull extended from 15 to 17 cm. back from the nasion and 10 cm. to the right of the middle line. The total nasion-inion line measured 34 cm. (fig. 3).

The wound healed completely at the beginning of April, 1916.

My observations were made at various times between January 2 and July 22, 1916.

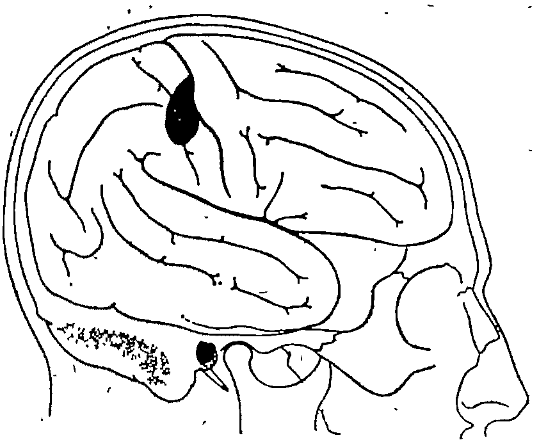

FIG. 5 (Case 3).

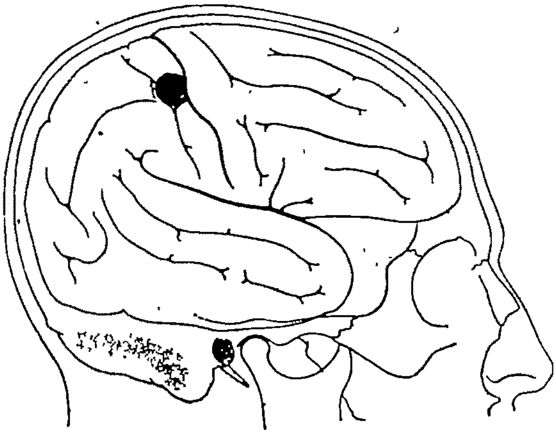

FIG. 6 (Case 4).

Case 2.—Lieutenant F. W. B., aged 31. Fully reported on p. 202 (fig. 4).

Case 3.—Bombardier J. W. B., aged 26. Fully reported on p. 206 (fig. 5).

Case 4.—Private F. B., aged 18. Wounded January 7, 1916, by a shrapnel bullet. No helmet.

The note which came with him said: "Compound fracture of the skull. On January 8, 1916, the edges of the long ragged scalp wound were excised.

Usual trephine operation to enlarge opening and remove depressed bone. Brain substance was oozing through. Blood-clot and brain washed away with a gentle stream of eusol."

"Had hernia cerebri, which disappeared under eusol dressings. At first there was complete paralysis of the left arm and leg. Now recovered as to coarse movements; fine movements still lack co-ordination. The ulnar fingers show most impairment."

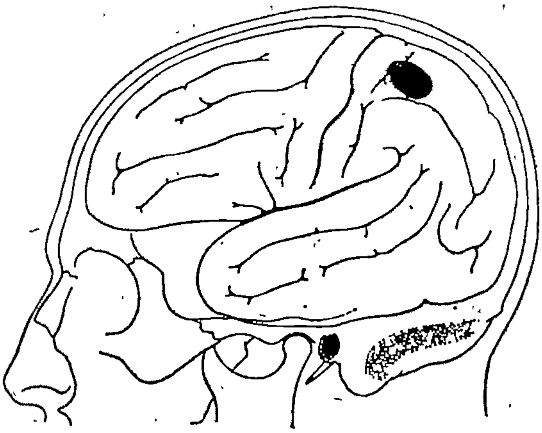

FIG. 7 (Case 5).

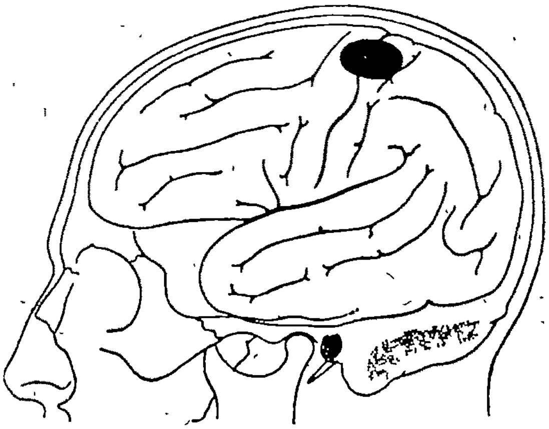

FIG. 8 (Case 7).

He was admitted to the London Hospital on April 4, 1916. The wound was healed and consisted of a crescentic scar on the right side of the head, extending from the anterior temporal to the posterior limit of the parietal region. The greater part of this was a scalp injury only; but in the centre of the wound the bone had been removed over an area 3 cm. in length horizontally and 1·2 cm. in breadth. Here the scar pulsated.

This area of removed bone extended from 18 cm. to 21 cm. back from the nasion. Its upper limit was exactly 5 cm. from the middle line. The total nasion-inion line measured 34 cm. (fig. 6).

My observations were made between May 9 and July 23, 1916.

Case 5.—2nd Lieutenant R. S. C., aged 28. Fully reported on p. 210 (fig. 7).

Case 7.—Lieutenant H. J. C., aged 39. Wounded July 17, 1915, by a rifle bullet. No helmet.

He was admitted to the Empire Hospital for Officers on October 27, 1915. No notes of any kind.

On July 18, 1915, some operation was performed, and he said he was trephined.

When he entered the Empire Hospital there was a long irregular scar on the left side of the head; in the centre was a minute granulating area, which was situated over the small trephine opening in the skull. This opening was oval in shape and measured about 2·5 cm. by 1·5 cm. in the two diameters. It was situated 18 cm. back from the nasion and 2 cm. from the middle line. The nasion-inion line measured 35·5 cm. (fig. 8).

On November 10, 1915, Mr. Walton removed two small pieces of bone, which lay in the brain substance about 1·5 cm. from the surface, but entirely closed off. The dura was intact except for the sinus.

The wound healed on December 23, 1915.

Exactly a year later the wound was found to be covered with a minute crust, and on November 18, 1916, Mr. Walton removed three small pieces of bone from the fibrous tissue on the surface of the brain. The wound healed finally November 29, 1916.

In this case the foot was most severely affected, and the injury had produced a definite right hemiplegia. Not only did the right plantar give an upward response, but the left was at first also abnormal in the direction of the movement.

The little finger showed more severe sensory changes than any other part of the hand.

Case 8.—Lieutenant L. A. D., aged 20. Fully reported on p. 221 (fig. 9).

Case 10.—Private C. H. E., aged 23.

Wounded at 6 p.m. on March 2, 1917, by a rifle bullet. He was wearing a helmet.

He says he was placed on the operating table in the early hours of March 3, 1917. No notes of any kind.

On March 6, 1917, he arrived at Rouen. The notes are as follows: "No notes, but trephine opening in the right parietal region. Small entrance wound, apparently not touched, in centre of flap. Large fluctuating hernia under flap, presumably large decompression."

He was admitted to the London Hospital on March 15, 1917. The incision and entrance wound had healed.

The scar of entry was situated from 18 to 20 cm. back from the nasion and 11 cm. to the right of the middle line. It measured 2 cm. in length horizontally and 0·75 cm. in breadth. Beneath it lay an irregular opening in the bone about 4·5 cm. by 2·5 cm. The nasion-inion line measured 35 cm. (fig. 10).

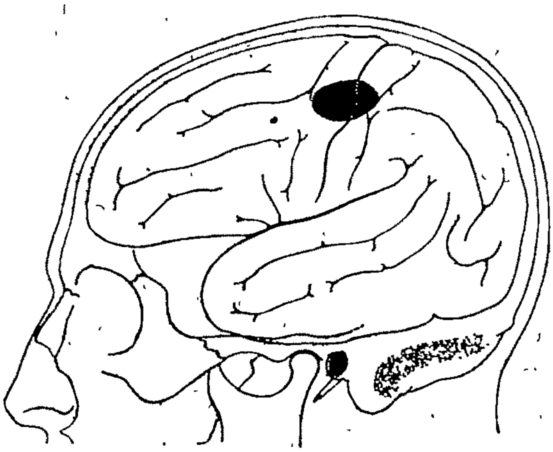

FIG. 9 (Case 8).

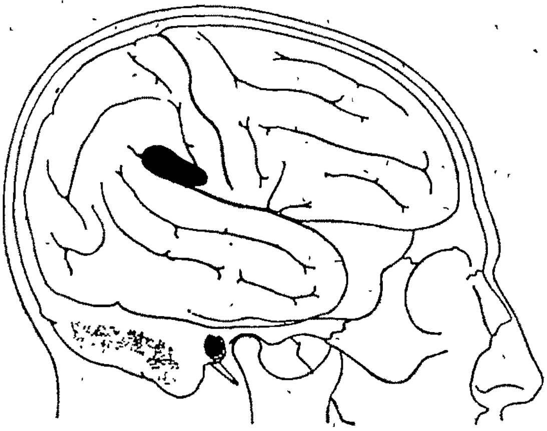

FIG. 10 (Case 10).

Case 11.—Lieutenant J. P. S., aged 29.
Wounded on May 3, 1918, by a rifle bullet. He was wearing a helmet.
On May 4 the wound was excised but he was not trephined.
No further operation.

Admitted to the London Hospital May 9, and was transferred to my care June 26, 1918.

The scar was an irregular incision in the left parietal region 8·5 cm. in length. It reached in front to within 14·5 cm. of the nasion, and posteriorly to 23 cm. back from the same point. The scar was throughout 8 cm. from the middle line. The bullet had evidently travelled from behind forwards. The total nasion-inion line measured 35 cm. (fig. 11).

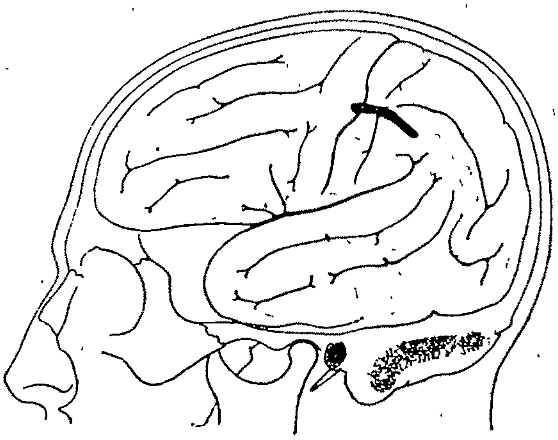

FIG. 11 (Case 11).

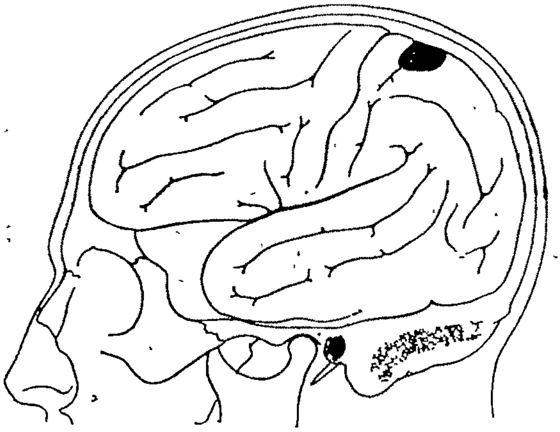

FIG. 12 (Case 12).

A radiograph showed no fracture of the skull.

This was a case of injury to the cortex without penetration of the skull.

The condition of the right hand was in every way characteristic. The maximum affection lay over the thumb and index finger. The index finger was definitely hypotonic.

Case 12.—Captain H. N. F., aged 22.

Wounded March 12, 1915, by a rifle bullet. No helmet.

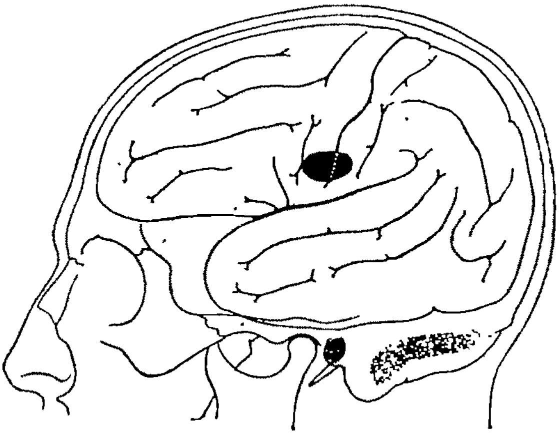

FIG. 13 (Case 13).

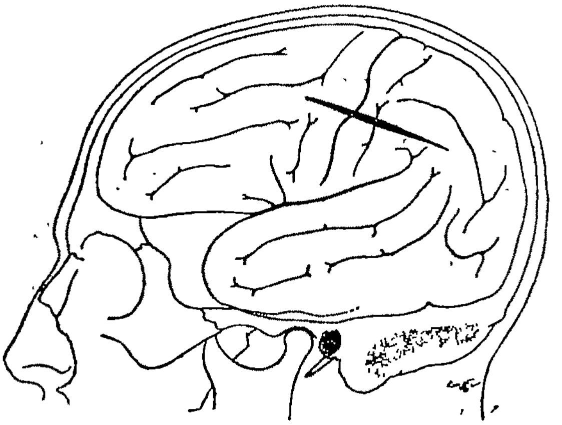

FIG. 14 (Case 14).

He was operated upon by Mr. Robert Milne on March 15, 1915. The skull was fractured and a small irregular trephine opening was made. The wound healed entirely in ten weeks. All headaches disappeared by the middle of April.

The leg had never been affected and two months after he was wounded the hand and arm had reached the condition in which I saw him.

He was brought to me by Mr. Milne in June, 1916, fifteen months after the injury. The wound was represented by a well-healed somewhat depressed scar, which did not pulsate unless he lowered his head; there was no pulsation in the erect position.

The opening in the bone was irregularly rhomboidal, 3 cm. in diameter from opposite angles. It was situated from 21·5 to 23·5 cm. back from the nasion, and the superior angle was 2 cm. from the middle line. The total nasion-inion line measured 35 cm. (fig. 12).

My observations were made between June 12 and June 25, 1916.

Case 13.—Lieutenant G. O. H., aged 21.

Wounded May 20, 1917, by a fragment of high-explosive shell. He was wearing a helmet.

At 9 p.m., May 20, "the gaping wound was excised. Depressed fragments removed. Dura open, discoloured and tense. Opened by a linear incision. Blood-clot and ruptured brain expressed."

On May 28, the note states, "Patient had motor aphasia and some slight impairment of the right hand."

When admitted to the London Hospital on June 14, there was a large area on the left half of the scalp covered with a network of healed plastic flaps. At a point in the centre of this labyrinth the skin was somewhat depressed and pulsated heavily. The site of the original wound could be seen, and it lay over a small irregular trephine opening. This was situated 16 cm. back from the nasion, and 5 cm. to the left of the middle line. It was roughly oval, 3 by 2 cm. in diameter. He had a tall head with a short posterior portion, and the total nasion-inion line measured 34 cm. (fig. 13).

Case 14.—Lieutenant E. C. H., aged 29.

Wounded at about 4.30 p.m. on October 1, 1916, by a rifle bullet. He was wearing a helmet.

The right hand was affected, and the right side of the face was numb. The leg was not affected at any time.

He was operated upon the same day, but was told "Nothing was found in the wound, which had not touched the bone."

He was admitted to the London Hospital on October 6, 1916, with a healed longitudinal scar of a surgical incision in the temporal and parietal region. There was no defect of bone. The anterior end of this scar was 13·5 cm. back from the nasion, the posterior end 23 cm. It lay as a straight line, somewhat obliquely to the middle line of the scalp: the anterior end was 6 cm., the posterior 9 cm. to the left. Its total length was 9·5 cm. The nasion-inion line measured 35 cm. (fig. 14).

The maximum affection of sensibility was found in the thumb and index finger. The index finger was somewhat hypotonic. Speech was affected at first. The lower extremity was in every way normal.

Case 15.—Lieutenant E. D. J., aged 22. Fully reported on p. 225 (fig. 15).

Case 16.—Private W. J., aged 38.

Wounded February 23, 1917, probably by a shell fragment. He was sniping behind a steel plate when it was hit.

The interchange of information card sent out by the Medical Research Committee stated: "Date of wound February 23. Could be roused to answer questions. Headache, vomiting, pain in eyes. Paresis and paræsthesia of left arm. Operation under local anæsthesia. Linear fracture of right parietal. Inner table fractured. Small laceration of dura. Active bleeding from vessel in dura. Bleeding controlled. Salt cigarette drain."

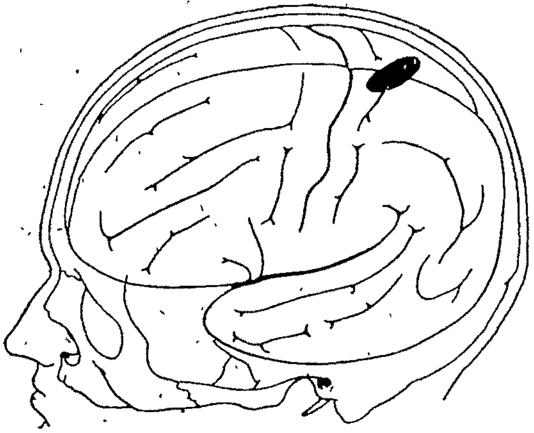

FIG. 15 (Case 15).

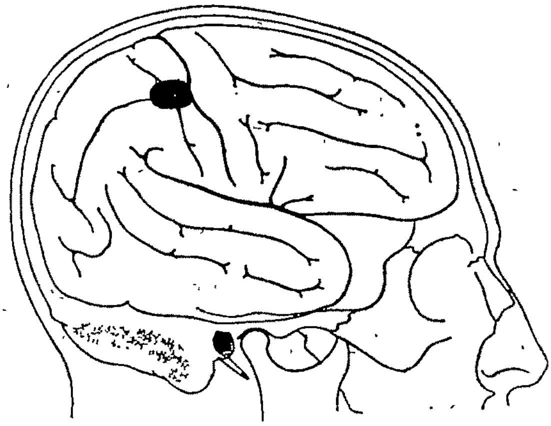

FIG. 16 (Case 16).

"March 17, 1917, paræsthesia all gone. Still paresis of fingers. Headache much better."

He was admitted to the London Hospital on March 22, 1917. There was a deep, pitted wound in the right parietal region, roughly triangular in shape.

No formal operation had been carried out and the loss of bone was extremely small. It measured about 3 cm. by 1 cm., and was situated from 18 to 20 cm. back from the nasion and 5 cm. to the right of the middle line. The total nasion-inion line measured 35 cm. (fig. 16).

The wound healed by May 8, 1917.

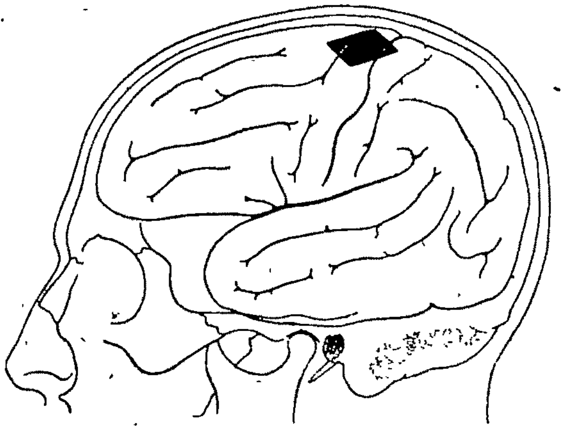

FIG. 17 (Case 17).

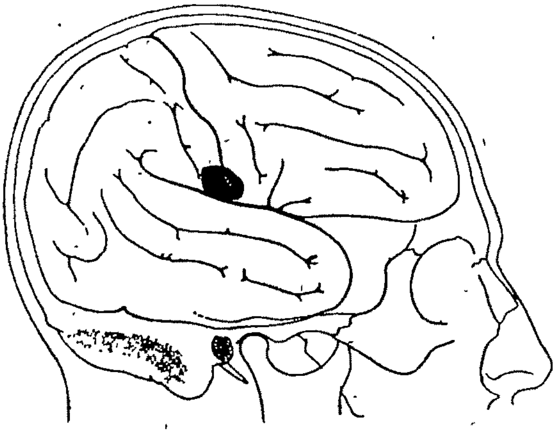

FIG. 18 (Case 18).

My observations were made between April 27, and June 4, 1917.

The remarkable condition in this case was the widespread sensory disturbance of the whole of the left hand; but tactile, thermal and painful sensibility were entirely unaffected. The loss of sensation to the tests for weight and form was particularly gross.

Case 17.—Private F. L., aged 29. Fully reported on p. 98 (fig. 17).
Case 18.—Colonel M., aged 46. Fully reported on p. 228 (fig. 18).
Case 19.—2nd Lieut. D. P., aged 23. Fully reported on p. 231 (fig. 19).
Case 20.—Private J. G. S., aged 21.

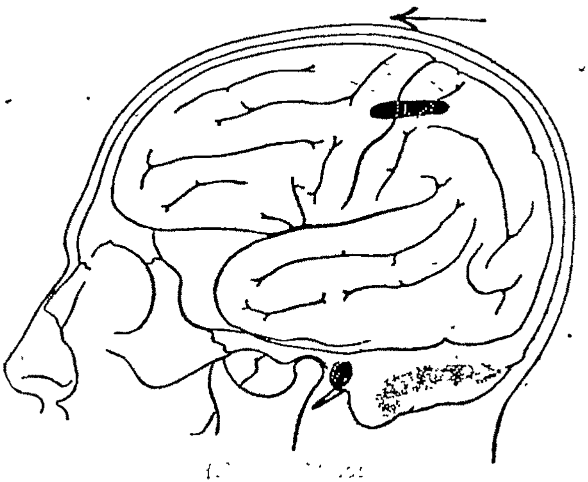

FIG. 19 (Case 19).

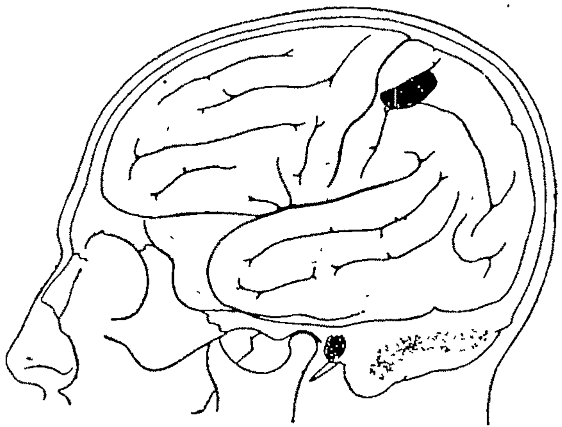

FIG. 20 (Case 20).

Wounded on July 18, 1917, by a fragment of a high-explosive shell. He was wearing a helmet.

Operated upon at 11 a.m. the same day. "Fracture of the skull. Dura not penetrated. Wound excised. Large fracture. All loose fragments of inner and outer table removed and bone chipped round evenly. No gross injury to dura."

July 24, 1917, arrived at the base by barge. "Had convulsions. Slow pulse. Motor aphasia. No optic neuritis. Slow cerebration. At 9 p.m. wound explored and a quantity of serum evacuated. Convulsions at 11 p.m. Lost use of right arm and leg."

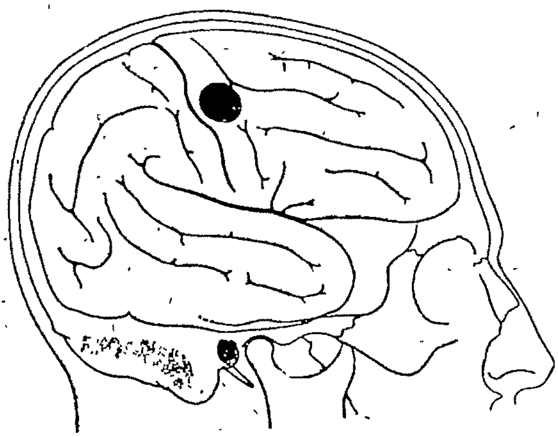

FIG. 21 (Case 21).

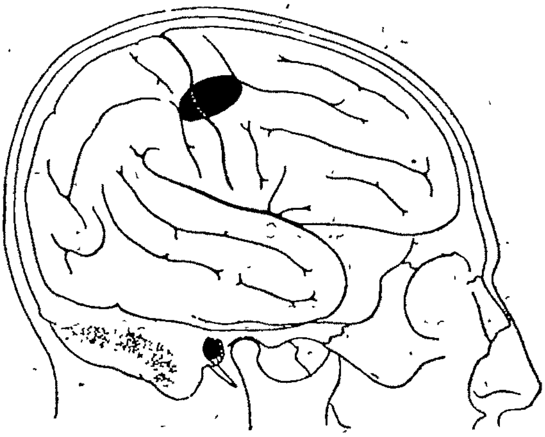

FIG. 22 (Case 22).

"July 26, more fluid evacuated."

"July 27, use of shoulder, elbow and wrist recovered in this order. Use of leg returned, toes last. Abdominal reflex returned. Motor aphasia has cleared up."

When admitted to the London Hospital on August 8, 1917, with a scar in the left parietal region, which was healed except for a small sinus discharging clear fluid. This healed entirely on August 17.

There was an irregularly oval opening in the bone 4·25 cm. in largest diameter. It was situated 20 cm. back from the nasion and was 5 cm. to the left of the middle line. A radiograph showed a definite removal of bone with smooth edges. The total nasion-inion line measured 34 cm. (fig. 20).

All the digits were affected, but the grossest loss was in the little, ring, and middle fingers.

Case 21.—Private J. H. T., aged 24.

Wounded on June 16, 1915, by shrapnel. No helmets at this time.

He was admitted to the London Hospital on July 8, 1915, with no notes of any kind. A large flap had evidently been turned down and in the centre the site of the original wound was obvious as a ragged hole. This was from 15 to 18 cm. back from the nasion and 8 cm. to the right of the middle line. The patch was 3 by 2 cm. and was pulsating. The total nasion-inion line measured 34·5 cm. (fig. 21).

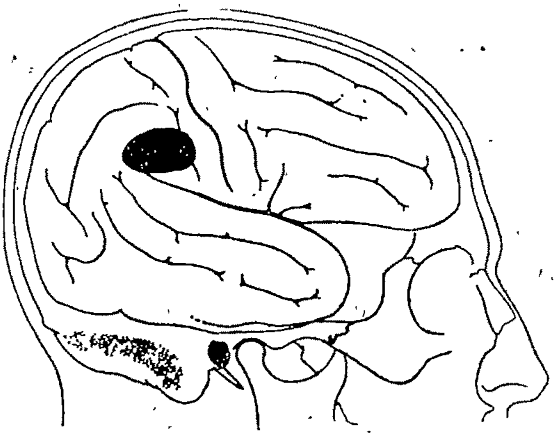

FIG. 23 (Case 23).

Case 22.—Corporal H. U., aged 29.

Wounded September 25, 1917, by shrapnel. No helmets at this time.

The same day a piece of shell casing and a shrapnel bullet were extracted.

The wound healed in the early part of December, 1915.

He came under my care on May 15, 1916.

There was an irregular rectangular removal of bone 4 by 1·5 cm. in the right parietal region. It extended from 16 to 20 cm. back from the nasion. The anterior edge was 4·5 cm. and the posterior edge 8 cm. to the right of the middle line. It was thus placed somewhat obliquely and pulsated in the centre. The total nasion-inion measured 34 cm. (fig. 22).

In this case the index finger showed the grossest sensory changes.

Case 23.—Private J. H. W., aged 22.

Fully reported on p. 235 (fig. 23).

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EDITOR.

[NOVEMBER, 1918.]

BRAIN.

PARTS III and IV, VOL. 41.

THE OCCURRENCE OF UNSUSPECTED INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM IN UNSELECTED CASES OF SYPHILIS.

BY PAUL FILDES,[1] RODERICK J. G. PARNELL, AND H. B. MAITLAND.

(From the Section for Venereal Diseases and from the Laboratory, Royal Naval Hospital, Haslar.)

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[1] Working in the Navy on behalf of the *Medical Research Committee.*

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I.—Introduction.

During recent years great interest has developed in the study of syphilis as it affects the central nervous system in the early stages of the disease.

It has been realized gradually that "gummatous meningitis," which a few years ago was the only recognized form of syphilitic brain disease, other than dementia paralytica, is an incorrect term if it implies that the lesion is a tertiary phenomenon only.

It became quite common to find this condition in the later secondary stages and then earlier in the secondary stage. Next, cases of "secondary syphilitic meningitis" were quoted which could not be accurately described as "gummatous" at all.

With increasing investigation of early cases of syphilis by neurologists, it began to be suspected that affections of the nervous system were really not uncommon in such patients, although they did not necessarily lead to pronounced symptoms.

Evidence then began to accumulate that such affections as were clinically detectable were in reality only a fraction of those which really existed; that the nervous system was frequently attacked without any outward indication of this being manifest.

The origin of this further advance may be taken to be a series of papers by Ravaut (1903) [19]. This author performed the operation of lumbar puncture upon a number of secondary syphilitics, most of whom showed no evidence of nervous disease, and in a large proportion found a pleocytosis and other pathological changes to be present in the cerebrospinal fluid. For instance, in 100 cases of early secondary syphilis there were only twenty-eight which could be said to have quite normal cerebrospinal fluids.

After this observation no further contribution was made to this subject for several years.

About the year 1911 a discussion arose as to the effect of "606" treatment upon the central nervous system. It was asserted by one school, and especially by Finger, that "606" actually induced a toxic reaction in the central nervous system which was recognized by an increased number of nervous signs following the treatment.

The nature of this toxic reaction was not at first clear. Some considered it to be a direct arsenical intoxication such as was known to occur (optic atrophy) after the administration of arsenic, while others considered that the lesion was syphilitic. This latter view soon became general (*vide* McIntosh and Fildes, 1911 [16]), and then the question arose as to whether these syphilitic affections of the central nervous system were commoner after "606" than after mercury. An extensive literature appeared upon this subject of the "nerve relapse." Ehrlich and his school argued that such relapses were quite common in the literature of mercury treatment (Benario). Without entering into all details it may be said that at the present time it is accepted that this was the case and that nervous affections are not unduly common after "606." On the other hand it is also accepted that such manifestations are remarkably common not only after "606" but before "606" or any other treatment has been administered.

The recognition of these "nerve relapses" and the consequent discussion greatly stimulated the neurological examination of cases of syphilis. Small signs were recognized which were formerly missed and the operation of lumbar puncture became a routine in neurological clinics. It was however only the cases which exhibited nervous signs which came into the hands of the neurologist, and thus it only gradually became evident that changes might be present in the cerebrospinal fluid when no obvious nervous lesion existed, as had been shown already by Ravaut.

These observations are sparse and often trivial.

In 1911 Boas and Lind [4] examined six cases of untreated secondary syphilis and found a slight increase in cells in two. Fraenkel [11] in 1912, in fifteen cases of syphilis described without detail, but including some late secondary cases, found five with increased cells and positive Wassermann reactions in the fluid. Whether the five cases on careful examination would have presented signs of nervous disease is not clear. The remarks upon the nervous system are confined to the letters "O.B."

Zaloziecki and Frühwald [24] in the same year examined thirty cases of early but unspecified syphilis without evidence of nervous disease. Four of these had an increase of cells in the cerebrospinal fluid (11 to 24 per cubic millimetre), five had 22 to 87 cells and also increased globulin, while two had 94 and 212 cells, increased globulin and positive Wassermann reactions. One of these was a late case of secondary syphilis (infection two years), and ophthalmological examination revealed a "pseudoneuritis." Otherwise the nervous systems of both were normal.

Bergl and Klausner (1912) [5] examined twenty-six cases. Four of these were suffering from primary syphilis and two had eight cells per cubic millimetre. The remaining twenty-two were secondary syphilitics, and fifteen were described as showing "pleozytose," while one had a positive Wassermann reaction in the cerebrospinal fluid.

Altmann and Dreyfus (1913) [1] made the first important contribution to the subject. These authors described cases with cells in the cerebrospinal fluid up to 15 per cubic millimetre as having "slightly altered" cerebrospinal fluids. These are not included in the following table of their results. No neurological examinations were made.

Stage of syphilis	Number of cases	Number with Wassermann reaction positive in blood	Number with cells over 15	Number with Wassermann reaction positive in cerebrospinal fluid
Primary	8	2	0 (One with 13 cells) (One with 7 cells)	0
Secondary with chancre ..	21	21	5	0
Secondary without chancre	35	35	12	11
Tertiary	2	2	0	0
Latent	85	46	0	0
,, ...	19	19	10	10

With regard to the "latent" cases, all had been treated with mercury, but none presented themselves at the hospital for obvious syphilitic conditions. These authors also showed that the abnormality in the cerebrospinal fluid may be reduced by salvarsan treatment, although the number of cells may be temporarily increased after insufficient dosage.

In 1913 Ellis and Swift [6] published twenty-two cases of early untreated secondary syphilis. None of these showed any nervous lesions, but three had 12, 12 and 13 cells per cubic millimetre.

Gamper and Skutezky (1913) [12] also examined twenty-eight similar cases. Nine of these had over 10 cells per cubic millimetre in the cerebrospinal fluid, and of these, four had positive Wassermann reactions in the fluid.

In the same year Fildes and McIntosh [8] referred to twenty-three cases of syphilis, without signs of nervous disease, in whom the Wassermann reaction was negative in the cerebrospinal fluid. These included all stages of syphilis except the primary. As was pointed out, however, these and other cases had been subjected to an elaborate neurological examination by Dr. H. Head and Dr. Fearnsides, and signs of nervous disease were found in several similar cases, which were then transferred to another category. Thus these results were not opposed to the findings of others.

In order to obtain a correct estimate of these abnormalities it is necessary to make routine examinations of all cases without selection, and such appear to have been first carried out by Engman, Buhman, Gorham and Davis (1913) [7].

Among other examples, these authors examined thirty-six cases of syphilis up to two years after infection. Four of these had a slight increase in cells, while three had increased cells, globulin and positive Wassermann reactions in the fluid. Of these three, two had symptoms of syphilis and one had unequal pupils.

During the two following years Wile and Stokes published several papers upon this subject [20, 21, 22]. These authors also made routine examinations of all syphilitics and laid special stress upon the occurrence of "neuroretinitis" and diminished bone conduction. For instance, in six cases of primary syphilis they described no less than five instances of "neuroretinitis." We have tabulated the findings in these six cases of primary syphilis as a sample of the results obtained.

Number of case	Duration of primary lesion (weeks)	Eye	Ear	Nervous system	Cells in cerebrospinal fluid	Wassermann
1	6	Normal	Normal	Normal	Normal	Negative
2	5	Neuroretinitis	,,	,,	7	Very slight positive
3	5	,,	,,	,,	5	Positive
4	4	,,	"Involved"	Rombergism, pupils sluggish and unequal.	200	Negative
5	1	,,	Normal	Knee and Ankle-jerks increased.	15	Negative
6	?	,,	Diminished bone conduction	Normal	5	Negative

It may be pointed out that these phenomena are not discussed in detail.

Hauptmann (1914) [14] examined fifteen cases of primary and secondary syphilis and found no abnormalities except in one case of secondary syphilis. This showed increased reflexes and had sixty-nine cells with a negative Wassermann reaction in the cerebrospinal fluid.

Leopold (1914) [15] examined sixty-seven cases and found between 0 and 10 cells per cubic millimetre in forty-three and over 10 in eleven. The Wassermann reaction in the cerebrospinal fluid was positive in six of these. Routine neurological examinations were made and various slight abnormalities were found to be present.

Gutmann (1914) [13] examined fifty-seven cases of early syphilis and found one case of early secondary syphilis with 13 cells, and seven cases of secondary syphilis with increased cells, of whom two had a positive Wassermann reaction in the cerebrospinal fluid.

Audry and Lavau [2] also in the same year described a case of primary syphilis with 40 cells.

The most recent paper upon this subject, and also the largest series of cases, was published by With [23] in this Journal in 1918, although the paper was written two years earlier. His results may be tabulated as follows:—

Stage of syphilis	Number of cases	Number with positive Wassermann reaction in blood	Number with cells over 10	Number with Wassermann reaction positive in cerebrospinal fluid
Primary	45	0	1	1 (partial)
Primary	37	37	3	0
Secondary, early with rash, 3-5 months after infection	134	?	13	2
Secondary; later treated cases	41	?	4	1
Latent	41	41	4	2
Tertiary..	18	?	1	0
	316		26	6

Thus in the categories mentioned With found over 12 per cent. with a pathological increase in the cells in the cerebrospinal fluid. The whole of this author's series included 439 cases of syphilis, but those already quoted are the only ones which bear upon this subject.

It may be remarked that there is no clinical description of the cases,[1] and thus it is difficult to satisfy oneself on the instance of primary syphilis with a slight positive Wassermann reaction in the cerebrospinal fluid. The serum was negative and there were no cells. It is also not quite certain to what extent this series is a purely random selection.

It will be recognized that many of these papers which have been referred to are in several respects unsatisfactory and fragmentary.

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[1] This was furnished by the Author, but could not be printed owing to want of space,—EDITOR.

It has appeared to us that in order to emphasize the existence of an abnormality of the central nervous system, some writers have assumed a very narrow margin of safety. Several of the neurological abnormalities, for instance, are hardly definite enough to carry much weight in argument. Very much stress can hardly be laid upon a number of cells up to 10 per cubic millimetre in the cerebrospinal fluid. These findings may indeed be abnormal, but at the same time it appears to us to require more definite evidence before we accept without reserve Ravaut's statement that 62 per cent. of cases of secondary syphilis have abnormal central nervous systems.

Looking however at these findings as a whole, it is certain that a very considerable proportion of cases of early syphilis have *obviously* abnormal cerebrospinal fluids and a very much higher proportion *possibly* abnormal cerebrospinal fluids. It may also be said that very few of these show obvious signs of nervous disease such as would produce any disability, while many show no discoverable signs of disease at all.

The existence of these early and undeclared affections of the central nervous system in early syphilis is of the greatest importance. In 1914 McIntosh and Fildes [17] called particular attention to this subject in connection with the pathology of "parasyphilitic" affections. They argued that a tertiary syphilitic lesion such as a gumma was due to a recrudescence in a remnant of spirochætes which had remained dormant *in situ* since the generalization during the acute stage. As Lang had stated as long before as 1881, "an organ which is the seat of a gummatous lesion must have been affected in the early period of syphilis."

McIntosh and Fildes were engaged in showing that the lesions of "parasyphilis" differed in no essential particular from those of tertiary syphilis, and in order to complete the comparison it was necessary to show that the central nervous system, which later developed "parasyphilis," might readily have been affected in the early stages. They thus collected such evidence of nervous disease as was then to be found in the literature, and which has been referred to in this paper. They also noted that Hoffmann had demonstrated, by inoculation, *Spirochæta pallida* in the cerebrospinal fluid of an acute syphilitic, and called attention to the experimental work of Steiner, and Jacob and Weygandt, upon rabbit syphilis, which indicated that a lesion of the central nervous system was a common phenomenon in these animals soon after inoculation.

Clearly this subject has a very important bearing upon the treatment of syphilis.

If it is true that the infection of the central nervous system in the early stages may be the percursor of dementia paralytica in the later, it is important to determine the conditions under which this development takes place. The proportion of early cases which are affected is apparently large, and yet only a very small number of these develop dementia paralytica. The question thus arises, which of these early cases are likely to be affected in this way?

In ordinary tertiary syphilis there is reason to suppose that the patients so affected have remained actively syphilitic ever since the secondary stage, that they have had in fact more or less persistent positive Wassermann reactions. It is therefore legitimate to suggest provisionally that these cases, which subsequently develop dementia paralytica, have persistent reactions in their cerebrospinal fluids.

These matters are, however, merely speculative, and to be established a large number of observations are necessary over a period of years.

In order to study the subject more closely we have instituted an examination of the cerebrospinal fluid as a routine in all cases of syphilis, and hope to be able to follow some of these patients with a view to determining their subsequent fate.

II.—THE GENERAL NATURE OF THE MATERIAL, AND METHOD OF DEALING WITH IT.

At Haslar every man who enters the venereal section for syphilis or suspected syphilis has the full details of his case entered upon a recording card system, which is designed to save labour and to give all information on a systematic plan suitable for mass statistics. Every man has all applicable laboratory tests carried out as a routine.

The treatment of the cases also proceeds upon routine lines, particular courses being applied to cases of particular "type." We do not treat men as individuals, but as "types" according to a set routine. The basis of the treatment is a series of injections of "neosalvarsan compounds,"[1] carried out at short intervals.

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[1] Throughout this paper we refer to "neosalvarsan compounds" or "914," meaning any of the drugs of similar constitution, but different name, which are now upon the market. By "606" we mean salvarsan type drugs. Neither of these abbreviations include "galyl."

We have further instituted a system by which it is possible (if the exigencies of the Service permit) to follow the histories of all men after discharge to duty, and we hope that a sufficient number may be followed to throw important light upon the value of particular routine treatments in cases of a particular "type."

We do not give accessory forms of treatment, such as mercury, and endeavour to dissuade the medical officers in ships from giving it. We thus arrange a straight issue as to whether some five or six doses of a "neosalvarsan" drug are capable of curing a satisfactory percentage of men in the particular stage of the disease met with.

M. 207. (Established—March, 1918.) No : *H. R. 385.*

A—— B——. Age *18.* Rtng.: *O. Sea.* Ship : *Minotaur.* Type : *C 1.*

Adm. *May 1, '18* Disch. *May 20, '18* Days Sick, *19* Infect. Date, *Mar 25, '18.*
Place, *Portsmouth.*

S. Pallida		Wassermann		Clinical Condition
Date	Result	Date	Result	Original :—
May 2, '18	*Pos.*	*May 4, '18*	*Pos.*	Present :—*Multiple indurated sores on and under prepuce. Ulcer to right of frænum. General adenitis. Papulo-squamous syphilide, trunk and limbs.* Previous treatment :—*None.* Condition on discharge :—*Sores healed; rash fading.*

Treatment			Subsequent History				
Date	Dose	Drug.	Date for W.R.	Ship	Result	Date	Remarks
May 3, '18	*0·45 grm.*	*N.A.B.*	*Aug. 1918*	*R.N.B. (Po)*	*Neg.*	*Aug. 11, '18*	
,, 6, ,,	,,	,,	*Nov. ,,*	*Renown*	*Neg.*	*Nov. 16, ,,*	
,, 9, ,,	,,	,,	*Feb. 1919*	,,	*Pos.*	*Feb. 8, '19*	*Wassermann*—relapse. Recalled
,, 12, ,,	,,	,,	*May ,,*				
,, 15, ,,	,,	,,	*Sept. ,,*				
,, 18, ,,	,,	,,	*Feb. 1920*				
			Aug. ,,				

C.S.F., *May 3, 1918. 4 lympho. per c.mm. W.R. neg.*

M. D. G. 13881/18. P.T.O.

Having established this routine system of examination and treatment (*vide* Fildes and Parnell [10]), we determined to investigate in a large series of cases the existence of unsuspected nervous disease, such as has been recognized to exist in cases of syphilis during recent years. We hoped that if we were able to record the outstanding features of the state of the central system of every man, our "follow up" system might in the future throw important light upon the mechanism of the onset of the later nervous manifestations of syphilis.

We therefore instituted the system of examination described in the following pages with the assistance of colleagues who specialized in particular branches.

The preceding illustration of the obverse of the official card, filled up with a hypothetical case, will give an indication of the system of examination to which all men were subjected.

The number "H.R. 385" is the man's reference number, which is inscribed upon his syphilis papers which he carries with him. The letters denote that Haslar was the original treating hospital to which all subsequent specimens of blood for Wassermann tests must be sent. The "Type C. 1" indicates that the man was a "C." case—namely, secondary syphilis up to eighteen months from infection. The number "1" indicates the particular course of treatment applied. These "types" are used in the classification of the results of treatment. The date of infection is always recorded and, with judicious questions, can often be fixed accurately. The "treatment" column gives the treatment administered in the hospital. This example is the "1" course; the drug "N.A.B." is novarsenobillon.

The "subsequent history" section is of great importance. The "date for W.R." refers to the dates on which specimens of blood are to be sent in from ships and is automatically printed on the card with a rubber stamp. We allow for two years' observation.

In the "remarks" column we add recommendations which are sent with the report of the blood tests to the medical officers in the ships in which the man is then serving.

At the bottom of the card we add the results of the examination of the cerebrospinal fluid, and of the eyes, ears and nervous system when these are examined.

The reverse of the card shown opposite, deals with reinfections or relapses upon a similar plan.

We have made every effort to recall cases which have shown abnormalities of the central nervous system, for examination, but of course their return is dependent purely upon the exigencies of the Service. Upon discharge from hospital the men return to barracks, and from there they are drafted back to ships. It is almost impossible to send men back from the Fleet, and therefore we are forced to make a re-examination while they are still at the barracks, and the period between treatment and re-examination is thus shorter than we should otherwise require. It will be seen that we have been able to make this re-examination in a proportion only of the total abnormal cases.

Relapse or Reinfection.

Clinical Condition:

Treatment			Subsequent History				
Date	Dose	Drug	Date for W.R.	Ship	Result	Date	Remarks

It should be particularly understood that in this investigation we have included every man who entered the venereal section from the end of January, 1918, to May 31, 1918—namely, 624 different cases. This is of special importance, because the series is a purely random selection and the results therefore indicate what may be expected from a similar random selection made elsewhere on similar material. We have in no sense accepted cases or endeavoured to obtain cases which might show interesting or abnormal results upon examination. The few men who have exhibited obvious signs of nervous disease have entered the section in a *bona fide* manner; the great majority of these, particularly all those who would be diagnosed as "tabes" or "general paralysis," are, of course, admitted into the neurological section of the hospital, with which we have no direct concern.

III.—THE ROUTINE ADOPTED FOR THE EXAMINATION OF THE CENTRAL NERVOUS SYSTEM.

(1) *Classification of Cases.*

A classification of cases of syphilis into "stages," such as primary or secondary, does not convey to us any essential meaning, and therefore we have divided our material upon a time basis, or upon a basis which embraces a time and clinical factor. Thus our "division" of the cases is as follows:—

Division 1.—Cases in which *Spirochæta pallida* is found in an inoculation lesion, before the Wassermann reaction has become positive in the serum.

Division 2.—Cases, usually with an inoculation lesion, but with no other signs or history of general infection, except glandular enlargement and a positive Wassermann reaction, up to and including twenty-six weeks from the date of infection.

Division 3.—Cases with signs or history of general infection, including a positive Wassermann reaction up to and including eleven weeks from the date of infection.

Division 4.—Similar cases from twelve to twenty-six weeks inclusive after infection.

Division 5.—Similar cases over twenty-six weeks from infection.

Division 6.—Cases without signs other than a positive Wassermann reaction over twenty-six weeks from infection.

It will be observed that Divisions 1 and 2 include early and later cases of "primary" syphilis; Divisions 3 and 4 early and later cases of "secondary" syphilis; Division 5 still later cases of "secondary" and "tertiary" syphilis; while Division 6 includes "latent" cases.

It may be pointed out that some cases do not present conspicuous "primary" or "secondary" lesions, and thus could not be classified upon the ordinary basis of "stages." These we have dealt with purely upon a "time" basis.

It is sometimes particularly difficult to decide upon Division 2 or Division 6 when a man has merely a recently healed sore of uncertain nature, the date of infection with syphilis being then unobtainable.

We do not know whether such a man is a recent or an old case: whether he has not yet developed a more or less normal "rash," or whether he has passed the period when this should have appeared and is not going to develop one. In the first instance he should be in Division 2 and in the second in Division 6. The distinction between the two depends almost entirely upon the duration of the infection.

(2) *Lumbar Puncture.*

(*a*) *Technique.*—When lumbar puncture is made part of the routine examination of every case of syphilis, it is necessary that the operation should cause little discomfort to the patient. Especially is this so in the instances where repeated examinations of a pathological cerebrospinal fluid are required. By using a technique that is practically painless it is not difficult for the operator to secure the confidence of the patient.

The following is the technique we employ: The patient is placed in a sitting posture with the back bowed and shoulders drooped. The skin is prepared by rubbing with ether and spraying with iodine. The skin around the selected interspinous space is then anæsthetized by injecting a 0·5 per cent. solution of novocain through a fine needle until a circular blanched raised area is obtained. Lumbar puncture is then carried out with a Barker's needle 3½ in long; 2 c.c. only of fluid are removed, and the patient is kept in bed for the remainder of the day. As a rule, the first prick of the fine needle is the only painful sensation, and no after-effects are felt; but a few cases have headaches, a feeling of stiffness in the neck and back of the head, or malaise, for one or two days, persisting sometimes for a week. The exact cause of these symptoms we do not know, but they are probably the result of a disturbance in the cerebral circulation. In an endeavour to prevent them, for a time we injected, through the lumbar puncture needle, an amount of sterile physiological salt solution equal to the amount of fluid removed, but headaches were not lessened, so this was abandoned. The best prophylactic we have found is rest in bed for the remainder of the day, and it is our custom not to give intravenous treatment on the day lumbar puncture is performed.

(*b*) *The estimation of the number of cells in the cerebrospinal fluid.*—The cell count requires to be done accurately. We have used throughout the method of Baker [3]. The following is the technique: Calibrate a pipette holding about 0·5 c.c. into five equal volumes. Shake the tube of recently drawn cerebrospinal fluid. Take up four volumes and then one volume of polychrome methylene blue (diluted 1 in 3 with water) and discharge them into a small test tube or centrifuge tube. Allow to stand for a minute or two and then after shaking transfer a drop to a Thoma-Zeiss cell and cover in the usual way. Examine with a $\frac{1}{8}$-in. objective, and pull out the draw-tube until the diameter of the field is exactly eight of the smallest squares. Mark this point for future use. Count the cells in fifty fields with a mechanical stage and multiply this number by two. This will give the number of cells per cubic millimetre. If polymorphonuclear cells are seen the number is noted. If a small amount of blood is present, the number of red cells per cubic millimetre is estimated at the same time and one cell is deducted from the white cell count for every 1,000 red cells per cubic millimetre. This method is very accurate and rapid.

Some difficulty is always experienced in setting precise limits to the number of cells which may be considered normal. We have not ourselves examined a large number of normal persons to settle this point, but have taken the ordinarily accepted limits. Perfectly normal fluids do not in our opinion contain more than 2 or 3 cells per cubic millimetre; the majority indeed contain none, but we have taken up to 4 as being within physiological limits. We are not, however, prepared to assert that 6 cells is abnormal, because it is undesirable to over-estimate the number of pathological cases. We, therefore, have taken 10 cells as being an index of an abnormal condition, and in doing so follow a generally accepted practice. Those cases which have between 5 and 9 cells per cubic millimetre (both inclusive) we regard as "doubtful" and classify as such. At the same time we are of opinion that these "doubtful" cases are more likely to be abnormal than normal.

(*c*) *The Wassermann reaction in the cerebrospinal fluid and serum.*—The general technique adopted for carrying out this reaction is that of Fildes and McIntosh, described in this Journal [8]. Its later modification appears in the Interim Report upon the Wassermann Reaction by a Special Committee of the *Medical Research Committee*, upon the standardization of pathological methods [9]. The serum was always tested qualitatively in one dilution, while the cerebrospinal fluid was estimated quantitatively by the method of falling quantities of fluid. The largest amount of fluid used was 0·8 c.c. (tested with 0·5 c.c. of a 5 per cent. suspension of deposited sheep's corpuscles). The other quantities were 0·2 c.c., 0·05 c.c., and 0·012 c.c. The maximum quantity tested was four times that used in the work by McIntosh and Fildes, collaborating with Head and Fearnsides in this Journal [18]. The second dilution corresponds to the first in that investigation; the third with the third, and the fourth with the fifth. There is, however, a difference in the technique used in the two investigations. While in the first the fluid was tested *unheated*, in the present the fluid is inactivated in the same way as the serum. We have made this modification because of the large amount of cerebrospinal fluid used in the present case. It has been remarked already [8] that a demonstrable difference may often be noticed between the Wassermann reaction in the cerebrospinal fluid from a case of "central syphilis" (G.P.I.) and "meningo-vascular syphilis." The reacting substance in the latter is more thermolabile than in the former. It was suggested that this is due to the presence of a transudate from the blood in the acuter meningo-vascular lesion. That is to say, the reacting substances in the cerebrospinal fluid in the meningo-vascular lesion are derived from two sources—namely, the local diseased tissues and the blood. This blood may be a very disturbing factor if the fluid is unheated, since unheated serum, even from a non-syphilitic, is capable of giving a positive reaction in a high dilution, and when eight times as much fluid is used as serum in the test, the concentration of unheated serum would almost certainly be sufficient to affect the result in some cases.

In cases of early syphilitic meningitis we think that a considerable proportion of the reacting substance in the fluid is derived as a transudate from the blood, in the same way as a positive reaction is obtained in the case of syphilitics with meningococcal or tuberculous meningitis. If the fluid is tested in an unheated condition this serum transudate will give a much stronger reaction than it should if the man is syphilitic, or it may even give some inhibition if the man is non-syphilitic. Thus, with unheated fluid the transudate reaction is much exaggerated, and although more positive results may be obtained it appears to us that they will be less "specific" as a test of disease in the central nervous system.

The method of stating the results is the same as in the previous publications. The figure 4 which is an abbreviation of four plus signs (+ + + +) means that a complete inhibition of hæmolysis occurred with the quantity of test fluid to which the figure applied. Thus 4444 means, in the case of the cerebrospinal fluid, that complete inhibitions took place with 0·8 c.c.; 0·2 c.c.; 0·05 c.c.; and 0·012 c.c. of fluid. 4400 means that complete positive reactions took place with the first two quantities and negative with the last two. 4 - - - means that a complete reaction was obtained with the largest quantity, but the others were not tested. In the serum the figure 4 means that a positive reaction was obtained with the ordinary test quantity of serum (0·1 c.c.). When reactions are not complete in any one case, the figures, 3, 2 or 1 are used to denote the degree of inhibition in that tube. When the results of examinations of serum and cerebrospinal fluid are expressed together, they are printed in the form of a fraction. The serum takes the place of the numerator and the cerebrospinal fluid of the denominator, thus: 4/4400.

(*d*) *Globulin and albumen tests.*—No globulin or albumen tests have been carried out in the cerebrospinal fluid of these cases, because, in view of the pressure of work, we did not desire to cumber ourselves with any tests which did not convey to us any more information than we had already gathered from the cell-count and Wassermann reaction. We have no doubt that the globulin and albumen tests are of value when other tests cannot be done, but they have always appeared to us to be somewhat indecisive.

(3) *The Examination of the Eyes.*

The eye-grounds were examined by Staff-Surgeon R. J. E. Hanson, O.B.E., F.R.C.S.Ed., R.N.V.R. Owing to pressure of work in the Ophthalmic Department, all cases with abnormal cerebrospinal fluids could not be investigated. Cases were selected, preference being given to those with high cell counts. No control cases were necessary.

(4) *The Examination of the Ears.*

Examination of the ears was largely carried out in the Venereal Section, with the co-operation of Temporary Surgeon E. C. Dunlop, R.N. One hundred unselected cases were used as controls: These consisted of fifty syphilis cases with normal cerebrospinal fluids, and fifty non-syphilis cases (persons who averred that they had never had syphilis, and whose sera were negative). In all cases Weber's, Rinne's and Schwabach's tests were applied. The object of the examination was to determine the condition of the internal ears. These tests could not be applied as a routine in all cases of syphilis. They were selected, but not to such a marked extent as with the eye examinations.

(5) *Examination of the Nervous System.*

The examination of the nervous system adopted as a routine could not be an elaborate one. In nearly every case with a positive cerebrospinal fluid we investigated :—

(*a*) The functions of the cranial nerves, except for taste and smell.

(*b*) The reflexes: (i) Pupils—to light and in accommodation, (ii) knee-jerks, (iii) ankle-jerks, (iv) elbow-jerks, (v) abdominal reflex, (vi) cremasteric reflex, (vii) plantar reflex, (viii) palate reflex. The organic reflexes, the state of the sphincters, and the intellectual functions were examined only in special cases with clinical nerve lesions.

(*c*) Sensory functions: (i) Sense of touch and pressure, (ii) sense of pain. These were tested with cotton wool for epicritic sense, and the point of a pin for protopathic sense. The temperature sense was not tested.

(*d*) Co-ordination. Romberg's sign.

IV.—THE RESULTS OF THE EXAMINATION OF THE CENTRAL NERVOUS SYSTEM.

(1) *The Examination of the Cerebrospinal Fluid.*

(*a*) *General statement of the number of cases with abnormal cerebrospinal fluids.*—Six hundred and twenty-four cases of syphilis have been examined during this investigation; of these 115 exhibited a pleocytosis of 10 or more cells per cubic millimetre, while seventy-three came within the "doubtful" limit of 5 to 9 cells, both inclusive. That is to say, 18 per cent. of these cases were found to have a definite pleocytosis and 11 per cent. came within the "doubtful" limits. If, as seems justifiable, the "doubtfuls" are considered to be abnormal and are included with the "positives," it is seen that 30 per cent. of these men were abnormal in this respect. With regard to the Wassermann reaction, we found forty-one with positive reactions in the cerebrospinal fluid (6 per cent.). In all except one of these, the cells were also positive, and thus the total number of abnormal cases is increased by one only. The Wassermann reaction was not positive in any instance of "doubtful" pleocytosis.

(*b*) *The incidence of pleocytosis in the various divisions of the cases.*—When the abnormal cases in the different divisions are tabulated, it is seen that the incidence is greater in some than in others.

From this table it appears that a pleocytosis exists in a small number of cases of primary syphilis, even before the Wassermann reaction in the serum has become positive, and that the number gradually increases until what may be described as the later secondary period, when no less than 29 per cent. are affected. During the later stages (tertiary), with manifest signs, the number affected is considerably smaller, but in the "latent" period, when manifest signs are absent, there is a very marked increase in the number of cells. The "doubtful" pleocytosis does not show this variation according to the stage of the disease.

TABLE SHOWING THE INCIDENCE OF ABNORMALITY IN THE DIFFERENT DIVISIONS.

Division	Number of cases	Number with positive cells	Number with "doubtful" cells
1	71	2	9
2	168	15	17
3	107	18	11
4	85	25	11
5	78	14	11
6	115	41	14
Results expressed as percentage of cases.			
1		2	12
2		8	10
3		16	10
4		29	12
5		17	14
6		35	12

It is desirable to illustrate the degree of abnormality met with in these cases with examples; we have therefore selected the following instances in the various divisions.

Division 1.

Case 845.—Infected January, 1918. *S. pallida* positive, March 16, 1918. Examined March 17: Ulcer in coronal sulcus; general adenitis; 16 L., W.R. 0/0,[1] N. normal, B. normal, O. not examined, S. normal.

Case 1195.—Infected March 28, 1918. *S. pallida* positive, April 27. Received 0·3 grm. galyl on April 28 and 0·25 grm. on May 8. Examined

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[1] Throughout this paper for economy of space we use the following symbols:—

16 L. = 16 lymphocytes per c.mm.; 16 L. + 2 P. = 16 lymphocytes and 2 polymorphs.
0 = negative Wassermann reaction (W.R.), using the largest quantity of test fluid.
4 = positive W.R.; 3 or 2 incomplete positive W.R., using the largest quantity of test fluid.
4 - - -; 4444; 4440; 4400 or 4000 = positive W.R. in the cerebrospinal fluid, using the full quantity (0·8 c.c.) only, or 0·8 c.c., 0·2 c.c., 0·05 c.c., and 0·012, as the case may be. When the result of the examination of serum and cerebrospinal fluid is given together, it is expressed as a fraction thus: 4 (serum) /4400 (C.S.F.).
N = physical signs of nervous disease.
B = bone conduction or otological examination.
O = ophthalmological examination.
S = symptoms of nervous disease.
606 = salvarsan type compounds } other than galyl.
914 = neo-salvarsan type compounds } other than galyl.
— = no observation.

May 11: Indurated ulcer at meatus; general adenitis; 8 L., W.R. 0/0, N. normal, B. normal, O. not examined, S. normal.

Division 2.

Case 986.—Contracted syphilis in June, 1916. Re-infected March 7, 1918. Sore on penis two days later. *S. pallida* positive, March 19, 1918. Examined April 11, 1918: Scar of recent sore on prepuce; scar of old sore in coronal sulcus; general adenitis; 114 L. + 2 P., W.R. 0/0, N. normal, B. normal, O. normal, S. normal.

Case 838.—Infected December 26, 1917. Examination on March 16, 1918: Kidney-shaped ulcer at junction of penis and scrotum, left side; *S. pallida* positive; general adenitis, also gonorrhœa; 8 L., W.R. 4/0, N. normal, B. normal, O. not examined, S. not inquired into.

Division 3.

Case 1230.—Infected about the middle of March, 1918. Had 0·25 grm. galyl, May 8, 1918. Examined May 12, 1918: Scar of sore on prepuce; sore on under surface of prepuce near edge, causing phimosis; general adenitis; fading macular syphilide; 16 L., W.R. 4/0, N. normal, B. diminished on both sides, O. not examined, S. dizziness for ten days.

Case 1238.—Infected March 10, 1918. Examined May 12, 1918: Scars of numerous erosions on glans, fading papulo-macular syphilide, general adenitis, condylomata ani; iridocyclitis right and left; 8 L., W.R. 4/0, N. normal, B. diminished on both sides, O. iridocyclitis, S. none.

Division 4.

Case 251.—Infected September 29, 1917. Examined November 29, 1917: Indurated ulcer on prepuce and indurated sore on lower lip; *S. pallida* positive in penile and lip sores: general adenitis; papular syphilide; Wassermann positive; had 3·15 grm. of "914" between November 29, 1917, and January 12, 1918 (the course being interrupted between December 3 and January 3 for unavoidable reasons). Re-admitted on April 1, 1918, with the following signs of relapse: Scar of sore on lip vesicular and about to break down; healing sore at frænum which was destroyed; general adenitis; papular syphilide; 352 L + 10 P., W.R. 4/4400, N. normal, B. diminished in both ears, O. not examined, S. none.

Case 555.—Infected in September, 1917. Sore on penis and rash. Examined on February 1, 1918: Scar of above sore; headaches and occasional giddiness, when stooping down, for one month; 750 L. + 144 P., W.R. 4/4000, N. normal, B. not examined, O. abnormal (*vide* p. 286), S. as above.

Division 5.

Case 1274. Infected in 1903. History of penile sore only. In 1914 had ulcers on back of right calf. Examined May 15, 1918: Inguinal and axillary adenitis; healing gummatous syphilodermia on anterior and internal aspects of right leg; scars of serpiginous ulcers with pigmented edges on posterior aspect of right calf and outer aspect of left knee; 200 L., W.R. 4/4400, N. normal, B. diminished on both sides, O. abnormal (*vide* p. 287), S. none.

Case 1297.—Infection date uncertain. Gonorrhœa in 1913 and in 1914. Examined May 27, 1918: General adenitis; patches of late syphilodermia on both palms, right knee, right thigh and alæ nasi; scars of recent ulcers on fauces; 8 L., W.R. 4/0, N. normal, B. diminished on both sides, O. not examined; S. none.

Division 6.

Case 825.—Infected 1903. Had penile sore, rash and sore throat. Examined March 13, 1918: Scar of sore on glans; scars of more recent ulcers on penis; 26 L., W.R. 4/0, N. abnormal (*vide* p. 282), B. not examined, O. not examined, S. present (*vide* p. 282).

Case 1175.—Infected September, 1917. Had sores under prepuce. Had two doses of galyl March 26 and April 5. Examined May 9, 1918; Scars of recent sores on prepuce; 8 L., W.R. 4/0, N. normal, B. normal, O. not examined, S. none.

Case 1164.—Infected September, 1917. Had penile sore, rash, sore throat. Had two injections of galyl March 9 and April 9, 1918, also three injections of mercury. Examined May 6, 1918: General adenitis; fading macular syphilide on both forearms; 1100 L., W.R. 4/4410. Had 4·65 grm. "914" between May 8 and June 17, 1918. After having had two doses of 0·45 grm. at three-day intervals, paralysis of the right sixth cranial nerve developed, but the treatment was not interrupted. June 20, 1918: 30 L., W.R. 0/4000. Had 1·8 grm. "914" between June 24 and July 8. July 16, 1918: 30 L., W.R. 0/3000. Paralysis of sixth nerve not completely resolved, but much improved.

(*c*) *The question of previous treatment affecting results.*—It has been suggested from time to time that the pleocytosis which had been recognized in these early cases of syphilis was associated with salvarsan or arsenical treatment. Finger, for instance, suggested that salvarsan had a direct toxic action upon the central nervous system, while others held, and still hold in some cases, that salvarsan predisposed to syphilitic affections of the central nervous system, and thus pleocytosis was common after this treatment as a "provocative" effect. In order to estimate the importance of this factor we have divided our cases into "untreated" and "recently treated." We have only taken those cases treated with an arsenical compound within fourteen days as "recently treated," since this is the period in which the "provocative reaction" is best detected in the serum or cerebrospinal fluid in the case of the Wassermann reaction.

TABLE SHOWING THE ABNORMALITY IN UNTREATED AND RECENTLY TREATED CASES.

Untreated.

Division	Number of cases	Number with pleocytosis	Number with "doubtful" pleocytosis
1	43	1	5
2	132	13	16
3	96	14	18
4	67	18	9
5	47	5	6
6	55	19	6
	440	70 (= 15 per cent.)	60 (= 13 per cent.)

Recently Treated.

Division	Number of cases	Number with pleocytosis	Number with "doubtful" pleocytosis
1	24	1	3
2	24	1	1
3	10	3	2
4	8	3	2
5	6	3	1
6	6	1	1
	78	12 (= 15 per cent.)	10 (= 12 per cent.)

From this table it is clear that arsenical treatment carried out during the period most satisfactory for inducing provocative reactions has no such effect. The incidence of abnormality is almost exactly the same in the untreated and recently treated cases. Thus we may conclude that this abnormality is in no way associated with previous arsenical treatment. On the other hand, if our results are compared with those of some others and found to be less abnormal, it may be objected that treatment had reduced the number of abnormal reactions. Few of our cases had, however, received any extensive course of treatment with arsenical compounds before coming under observation, but nevertheless we tabulate them to show the importance of this fact or factor.

TABLE SHOWING THE EFFECT OF TREATMENT (NOT RECENT) UPON THE INCIDENCE OF ABNORMALITY.

Cases treated, but not within fourteen days.

Division	Number of cases	Number with pleocytosis	Number with "doubtful" pleocytosis
1	4	0	1
2	13	2	0
3	1	1	0
4	10	4	0
5	25	6	4
6	53	20	7
	106	33 (= 31 per cent.)	12 (= 11 per cent.)

The untreated cases shown in the table on this page had 15 per cent. with pleocytosis, and 13 per cent. with "doubtful" pleocytosis; thus the treated cases exhibited a considerably greater degree of abnormality. This result may be taken as confirming the view that cases after salvarsan treatment show a greater incidence of nervous disease than cases after no treatment; but this would be an incorrect deduction. The high percentage affected in the treated cases is due to the large proportion of Division 6 cases which appear in the list in the above table. Taking Division 6 alone, there is no greater incidence of nervous diseases in the treated than in the untreated.

The reason for the large proportion of treated Division 6 cases is no doubt that these men, having been formerly admitted to a hospital for treatment, have come under the Naval "follow up" routine, and thus had Wassermann reactions carried out more often than men who had never been to hospital. Then, being found with positive reactions after the almost invariably inefficient treatment, they were again admitted for examination.

In general we conclude that the amount of treatment which our patients received had no effect upon our results one way or another.

(*d*) *The incidence of a positive Wassermann reaction in the cerebrospinal fluid in the various divisions of the cases.*—It is of importance to determine the stage of the disease in which the Wassermann reaction is most commonly present and at which the nervous lesion first becomes sufficiently pronounced to establish a positive reaction in the cerebrospinal fluid.

TABLE SHOWING THE INCIDENCE OF POSITIVE WASSERMANN REACTIONS IN THE CEREBROSPINAL FLUID IN THE VARIOUS DIVISIONS OF THE DISEASE.

Division	Number of cases	Number with positive W.R.	Number per cent with positive W R.
1	71	0	0
2	168	1	0
3	107	3	2
4	85	7	8
5	78	5	6
6	115	26	22

In this table it is seen that the positive reaction is not observed in primary syphilis until the Wassermann reaction in the serum has become positive (Division 2), but even then there is only one instance of a very slight reaction.

Case 513.—Infected September, 1917. Examined January 25, 1918. Indurated scar of recent sore on dorsum of penis; general adenitis; 84 L., W.R. 4/2000, N. not examined, B. not examined, O. positive (*vide* p. 285), S. not examined.

In the course of the routine clinical examination of cases, three men were placed in Division 2 who have since been transferred to Division 6. These three men had positive Wassermann reactions in the cerebrospinal fluid. We give here the details in order to justify ourselves and illustrate the difficulties of diagnosis.

Case 1130.—Mercantile Marine stoker, aged 38. Infected "during the last six weeks." Had a sore on the penis sixteen years before. Examined May 4, 1918: Indurated subpreputial ulcer dorsally; general adenitis; gonorrhœa; no *S. pallida* found on three occasions, but antiseptics had been applied; 18 L., W.R. 4/4- - -, N. normal, B. normal, O. not examined, S. general malaise for ten days before admission.

Case 1281.—Chief Petty Officer, aged 42. Said to have been infected March 10, 1918. Examined April 23, 1918: scars of papules on glans; slight general adenitis; 12 L., W.R. 4/4- - -, N. normal, B. normal, O. positive (*vide* p. 288), S. normal.

Case 1059.—Stoker, aged 23. Infected July, 1915 (primary syphilis). W.R. positive in August and December. 1915; negative in June, 1916. Treated with mercury and one dose of "606." (?) Reinfected December 15, 1917. (?) *S. pallida* found February 26, 1918. Examined April 20, 1918: Acquired phimosis; balanitis: no gonorrhœa; subpreputial induration; general adenitis; 42 L., W.R. 4/4400, N. normal, B. normal, O. positive (*vide* p. 287), S. normal.

These three cases had penile sores, and the question to be decided was, whether these were primary syphilis or non-syphilitic lesions in old syphilitics. The spirochæte test should be the deciding factor in such cases, but in one (Case 1281) the lesions had healed, in another (Case 1130) *S. pallida* could not be found, while in the third they were said to have been found outside this hospital, but we are not satisfied that this diagnosis was correct. This man had phimosis and balanitis with a thick yellow discharge, incorrectly diagnosed as gonorrhœa. Under such circumstances the differential diagnosis of *S. pallida* from *S. refringens* may be extremely difficult, and we have frequently known mistakes to have been made.

In the absence of the spirochæte test we may have recourse to clinical examination, and in only one was there serious clinical evidence of primary syphilis—namely, an "indurated subpreputial ulcer dorsally"; but we may remark that too much diagnostic value should not be placed upon "induration."

As regards evidence to establish the fact of these men being old syphilitics, this is forthcoming in two. One undoubtedly had syphilis shortly before, while another stated that he had a sore sixteen years ago. Two of the men were old seamen, and were of an age which suggests an older infection.

Our attitude towards these cases is that there is no certain evidence that they belong to Division 2, and if the evidence is fairly equally balanced we prefer to transfer them to Division 6, because otherwise they would be the only examples of a fully positive Wassermann reaction in the earlier division; such pronounced nerve lesions and eye lesions in a case of primary syphilis would be so remarkable that they should be established beyond a doubt before they are accepted.

In Division 3 the nervous lesion may be sufficiently advanced to give a few undoubted reactions in the cerebrospinal fluid.

Case 593.—Infected in November, 1917. Examined February 5, 1918. Scar of sore to right of frænum; marked general adenitis; both tonsils enlarged and covered with mucous patches: scars of recent condylomata ani. 740 L. + 2 P.; W.R. 4/4300, N. normal, B. normal, O. abnormal (*vide* p. 285), S. none.

Case 1258.—Infected early in April, 1918. Examined May 12, 1918. Scars of large ulcers on body of penis left. Two erosions, one on dorsum of glans left, and one subpreputial left. General adenitis; ulcers on tonsils: rhagades at left angle of mouth; follicular syphilide; 36 L., W.R. 4/4---, N. normal, B. positive, O. not examined, S. hard of hearing for several years, worse last month. Treatment, from May 18 to June 2, 2·7 grm. "914." Re-examined July 23, 1918: 0 L., W.R. 4/0.

Case 673.—Infected 1916. Primary syphilis treated with three doses of galyl. Reinfected January 29, 1918. Examined February 21, 1918: Small sore on penis to right of meatus; *S. pallida* present; condylomata ani; general adenitis; 82 L., W.R. 4/3000, N. not examined, B. not examined, O. normal, S. not inquired into.

In the later secondary period (Division 4) positive reactions are frequent, but rather less so in the manifest tertiary (Division 5).

The latent period (Division 6) shows the most remarkable result, no less than 22 per cent. of such cases being affected in this manner. We give here three ordinary examples:—

Case 829.—Infected May, 1914. Indurated sore at meatus. Treated with 0·3 grm. "606" in May, 1914, and three doses of galyl between July and September, 1917; also mercurial injections and pills. Examined March 17, 1918: No lesions; 98 L., W.R. 4/4400, N. normal, B. diminished right, O. abnormal (*vide* p. 287), S. not inquired into.

Case 520.—Infected May, 1916. Had two injections of galyl in May, 1916. and 0·75 grm. "914" in June, 1917. Examined January 25, 1918: General adenitis; 107 L., W.R. 4/4440, N. normal, B. diminished on right side, O. abnormal (*vide* p. 287), S. none. Had 4·5 grm. "914" between January 27 and March 10, 1918. On March 11, 1918: 16 L., W.R. -/4---. On June 8, 1918: 10 L., W.R. 0/0.

Case 740.—Infected November, 1916. Sore on penis, rash, sore throat. Had three injections of galyl, May and June, 1917, and twelve injections of mercury. Examined February 28, 1918: Scars of subpreputial sores; inguinal and cervical adenitis; 112 L.. W.R. 4/4000. Had 3·6 grm. "914" between February 28 and March 28 (four doses of 0·9 grm.). April 11, 1918: 10 L., W.R. -/3000, and 0·9 grm. "914" given. May 9, 1918: 10 L., W.R. 4/3000, and given 0·9 grm. "914" on the following day.

(*e*) *The relation of the degree of pleocytosis to the incidence of the Wassermann reaction in the cerebrospinal fluid.*—It is of interest to inquire whether a positive Wassermann reaction has any constant association with a particular degree of pleocytosis in the fluid. In the following table all cases with a positive Wassermann reaction are arranged with the number of cells and compared with the number of cases which gave a negative reaction.

Table showing the Relation of the Positive Wassermann Reaction in the Cerebrospinal Fluid to the Number of Cells.

Number of cells in the C.S.F.	Number of cases with W.R. pos.	Degree of reaction	Number of cases with W.R. 0
0—9	1	4--- (0 cells)	508
10—19	3	4--- (12 ,,) 4--- (14 ,,) 4--- (18 ,,)	36
20—49	4	4--- 4400 4--- 4---	26
50—99	11	4400 4400 4000 3000 2000 4--- 4400 4400 4100 4400 4400	6
100—149	7	4400 4400 4400 4400 4440 4000 4400	3
150—199	4	4400 4400 4400 4400	2
200—299	4	4400 4444 4--- 4000	0
300—499	2	4400 4400	1 (476 cells)
500—999	3	4300 4100 4---	0
1000—	3	4400 4--- 4410	0

This table shows that in 509 cases without pleocytosis only one was observed with a positive Wassermann reaction. That is to say, it is very uncommon to observe a positive reaction when there are no cells. This rule does not of course apply when dealing with ordinary cases of cerebrospinal syphilis, when they have been subjected to extensive treatment; but practically none of our cases had been so treated with the exception of this single instance in Division 5.

Case 88.—Infected 1907. Original symptoms and treatment not recorded. On November 9, 1917, admitted to hospital on account of a positive Wassermann reaction. General adenitis (old fibrous type) and scars of ulcers on both legs. Between November, 1917, and January, 1918, he received 2·7 grm. "914." On April 15, 1918, readmitted with bilateral eighth-nerve deafness; 0 L., W.R. 4/4---.

Among thirty-nine cases with a slight degree of pleocytosis (ten to nineteen cells) three were found with a positive Wassermann reaction. One of these (No. 627) was an old case of syphilis, and two were probably[1] old cases in the latent stage.

As the number of cells in the cerebrospinal fluid increases so do positive Wassermann reactions become more frequent. When there are over 200 cells per cubic millimetre a positive reaction appears to be almost constant. In thirteen instances only one had a negative reaction.

Case 1135.—Infected end of December, 1917. Examination May 4, 1918: Scars of multiple indurated small ulcers in coronal sulcus and on under surface of prepuce; *S. pallida* not found twice, but local antiseptics had been applied; general adenitis; gonorrhœa; 476 L., W.R. 3/0000, N. normal, B. normal, O. abnormal (*vide* p. 285), S. normal. One week later the Wassermann reaction in the serum was negative.

This case has been classed in Division 2, primary syphilis with a positive Wassermann reaction; but it is impossible to say whether this classification is correct, owing to the fatal practice of applying local antiseptics before spirochætes have been looked for.

It is clear from these results that positive reactions become more frequent as the number of cells increase. While only one in 509 is positive when there are between 0 and 9 cells, three out of three are positive when there are 1,000 cells.

(*f*) *The occurrence of S. pallida in the cerebrospinal fluid.*—In order to obtain final proof that the abnormalities of the cerebrospinal fluid were due to syphilis, several with the highest cell counts were examined for *S. pallida.* Thirteen of these contained over 200 cells per cubic millimetre, and the following technique was used: The 2 c.c. of fluid was placed in a small sterile glass tube, having a test tube end and a diameter of 1 cm. This was centrifuged at high speed for fifteen minutes. The fluid was then gently poured off, to be kept for a Wassermann test, leaving a deposit sticking to the bottom. Keeping the mouth of the tube downwards, the deposit was taken up in a capillary pipette with a rubber teat attached and placed under a cover-slip on a glass slide. It was then examined by dark-ground illumination. Fifteen cases were examined, in every instance within three hours after lumbar puncture, and in one only was *S. pallida* found. This man (Case 548, pp. 281 and 293) was infected twenty weeks previously, and had 1,000 cells in the cerebrospinal fluid.

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[1] *Vide* p. 277 for discussion of these two cases, Nos. 1180 and 1281.

(2) *The Results of the Examination of the Motor and Sensory Functions of the Central Nervous System.*

The number of cases which showed clinical evidence of abnormality of the central nervous system may conveniently be summarized by the following table :—

Division of disease	Number of cases with abnormal C.S.F.	Number of cases examined neurologically	Number of cases with clinical evidence of disease in the C.N.S.	Number of cases with symptoms	Number of cases with clinical signs
1	2	2	0	—	—
2	15	11	0	—	—
3	18	15	1	1	0
4	25	23	7	6	2
5	15	12	5	4	3
6	41	36	10	9	8

The details of the abnormalities detected in every case are as follows :—

Division 3.

Case 1230.—Dizziness for ten days.

Division 4.

Case 139.—Complained of weakness in his legs after walking.

Case 548.—For one month he had noticed that sometimes he was dizzy for a few moments after rising from a chair. He had been doing full duty in his ship until he came to hospital on account of his rash. He had no other symptoms, but his cerebrospinal fluid contained 1,000 cells per cubic millimetre.

Case 555.—For two months he had headaches and occasional giddiness after stooping down.

Case 560.—Weakness of facial muscles on left side.

Case 958.—Insomnia, nocturnal headache, vertigo.

Case 1056.—Complete bilateral paralysis of facial muscles. Weakness of the left external rectus muscle.

Case 1098.—Complained of unusual languidness.

Division 5.

Case 384.—Complained of diplopia for one month. He had ptosis of right eyelid.

Case 607.—Complained of headaches.

Case 840.—He had had spells of dizziness, during which he would have to support himself for a few moments; ankle-jerks absent.

Case 956.—Had difficulty in swallowing both fluids and solids for three months prior to entry. The throat was normal, and the symptoms disappeared completely after treatment.

Case 1071.—Paralysis of facial muscles on left side. Diminished sensation over area supplied by the fifth cranial nerve on the left side.

Division 6.

Case 125.—He complained of headaches and was very sleepy during the day. He easily got tired.

Case 627.—Headaches for three months before admission to hospital.

Case 753.—He had felt "drowsy" (the reason he reported sick) during the past six months.

Case 767.—For one year he had loss of memory, inability to concentrate, and was easily worried.

Case 825.—One year before admission he had numbness of the arms. On examination he had weakness of facial muscles on the left side.

Case 1064.—For four months has had frontal headaches and insomnia. Memory for recent events was poor.

Case 1084.—He complained of insomnia.

Case 1137.—For the past nine months he had attacks of unconsciousness and of severe vertigo lasting for several minutes.

Case 1141.—He complained of deafness for "women's voices."

Case 1142.—He complained of defective vision and diplopia. He had paralysis of left external rectus muscle.

The central nervous system was also examined in twenty-two cases in which the cerebrospinal fluid contained from 5 to 9 cells per cubic millimetre—i.e., "doubtful" cases. These included cases from each of the six divisions. In only one (Case 771, Division 3) was any abnormality found.

Case 771.—Infected December 18, 1917. Examined March 7, 1918: Balanitis: acquired phimosis; subpreputial sores; general adenitis; lenticular syphilide; 3 L., W.R. 4/0, N. normal, B. normal, O. not examined, S. occipital headache for nine days.

(3) *The Results of the Examination of the Function of the Internal Ears.*

The results of these examinations may be summarized as follows:—

Table showing the Number of Cases with Abnormalities of the Internal Ears. (Diminished Bone Conduction.)

Division	Number of cases with abnormal C.S.F.	Number of cases examined for internal ear function	Number of cases with internal ear function abnormal	Number of cases with bone conduction diminished on right side only	Number of cases with bone conduction diminished on left side only	Number of cases with bone conduction diminished on both sides
1	2	2	0	—	—	—
2	15	10	2	0	1	1
3	18	12	4	0	1	3
4	25	19	7	0	0	7
5	15	10	4	0	1	3
6	41	26	13	4	2	7

Of nineteen "doubtful" cases whose ears were examined six were found to be abnormal. Of these, two were in Division 2, and one in each of the other divisions, excepting Division 5. Thus, taking "positive" and "doubtful" cases together, thirty-six were found to be abnormal out of ninety-eight (36 per cent.).

As already stated, however, we determined to carry out a series of control examinations before drawing conclusions from these results. We first examined fifty men who stated that they had never had syphilis, showed no signs of syphilis, and gave a negative Wassermann reaction. Of these no less than fifteen showed diminished bone conduction, that is to say, a proportion practically as high as among the syphilitics. In six of these cases the abnormality was to be accounted for by gun fire, but among the others no sufficient cause was apparent.

We also examined fifty syphilitics with normal cerebrospinal fluids, and twenty-nine of these were found to have diminished bone conduction. Gun fire was probably responsible for the lesion in thirteen of the cases.

In view of the large number of "positives" among the controls, therefore, we are not prepared to lay much stress upon the value of this test and have now discontinued it.

(4) *The Results of the Examination of the Eyes.*

The following table shows the degree of abnormality met with during the ophthalmological examinations:—

TABLE SHOWING THE NUMBER OF CASES WITH ABNORMALITIES OF THE EYES.

Division	Number of cases with abnormal C.S.F.	Number of cases whose eyes were examined	Number of cases with abnormal eyes
1	2	0	—
2	15	3	3
3	18	8	5
4	25	18	9
5	15	7	6
6	41	22	17

No examination of the eyes was made in cases whose cerebrospinal fluid showed "doubtful" cells.

Staff-Surgeon Hanson has been able to recognize a number of extremely slight but none the less perfectly definite abnormalities in the eye grounds, which probably represent the same condition as that which Zaloziecki and Frühwald noticed and did not recognize, when they called it a "pseudoneuritis." Staff-Surgeon Hanson looks upon these abnormalities as representing early stages in the development of the syphilitic choroidal endarteritis and retinitis which is well known in later stages of the disease.

These early ocular changes contrast with those found in later stages with sectorial or insular scotomata the result of postchoroido-retinitic atrophy.

NOTES UPON THE OCULAR CONDITIONS, BY STAFF-SURGEON R. J. E. HANSON, R.N.V.R.

"*Retinitis ex Lue*" (*Early Stage*).

Typical signs.—The conditions vary in their intensity, but three types of signs in the fundus oculi were noted:—

(1) *Hyperæmia.*—The retinal venules especially were over-full.

(2) *Engorgement.*—Enlargement and cloudy appearance of the perivascular lymph spaces around the retinal vessels and around the papilla. (Luetic halo.)

(3) *Pigment overgrowth and disturbance of the retina.*—Small rounded woolly-looking opacities with fine spidery connective tissue changes, without apparent upset to the functions of the rods and cones.

In relation to No. 2 above, it is interesting to recall that there are physical and chemical changes in the cerebrospinal fluid, and that this is intimately connected with the subarachnoid contents, and these last with the retinal lymph-streams.

With regard to No. 3 above, one recalls the extensive pigmentation changes in the skin lesions of the secondary stage.

Summary.—There was little evidence of interference with function—i.e., with fields, form-sense, light difference, and colour thresholds. No alterations in the fundus oculi were observed in the cases seen shortly after the appearance of the inoculation lesion. In the later cases the changes in the fundus oculi were :—

(1) Symmetrical.

(2) Mainly peripheral.

(3) Diffuse and not circumscribed—i.e., not more marked nor intense at one part of the periphery than another.

No hyalitis nor choroiditis was observed but an underlying condition of choroidal endarteritis may be present in these cases of stippling of the fundus oculi.

The detailed reports of the examination of the eyes by Staff-Surgeon Hanson are given below :—

Division 1.

None examined.

Division 2.

Case 513.—Optic discs, no papillary œdema. Vessels of right and left are more distended than normal but not tortuous. Superficial fibres of retina at edge of disc are greyer than usual and less distinct (? increased tension in subarachnoid fluid). No hyalitis. Nothing specific in arterioles of retina.

Case 986.—(A reinfection case. He had syphilis two years previously and had been cured.) Right vision $\frac{6}{6}$, D. 0·5. Left vision, less than $\frac{6}{6}$, D. 0·5. Slight hypermetropia. Orthophoria. No opacities in media. Fundi: Right eye: disc, fovea and vessels normal, but there are some streaks of greyish-white appearance between the superior retinal vessels which are not of recent origin. These streaks after a short separate course toward the right disc appear to coalesce at about 1½ disc diameters from the disc. In right fundus also there are a few tiny areas of fatty degeneration about 0·5 mm. in diameter, approximately round. The retinal vessels are certainly not "plus." Immediately and completely and regularly around the right disc the retinal pigment is deficient over a zone 0·5 mm. wide. Left eye: disc, fovea and arterioles are normal. Venules are "plus." The grey streaks of right eye are here less obvious but still recognizable.

Case 1135.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Media clear. Fundi: Considerable pigmentation in excess in retina at periphery of right and left discs. The deeper portion of the retina shows tiny pale rounded areas (stippling). There is also an unusual amount of perivascular halo right and left.

Division 3.

Case 593.—No hyalitis. Discs right and left are a little hyperæmic and the surrounding retina is greyish in aspect. There is a trace left of perivascular lymph excess. No papillary œdema

Case 645.—There is retinal hyperæmia on the arterial side. Venules are normal. Discs and foveæ are normal.

Case 856.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Orthophoria. All details of discs are clearly seen, but there is some blurring at their temporal edges and in the adjacent superficial layer of the retina right and left. No general stippling. Vessels normal and symmetrical in size and contour.

Case 1119.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. D. 0·5. Colour perception normal. Fields normal. Orthophoria. Media and vitreous: clear right and left. Fundi: venules "plus." No enlargement of perivascular lymph sheaths noticeable. Stippling of right and left retina, especially in the periphery.

Case 1258.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Blurring of nasal disc margin. Cups clear at depth. No other changes noticed.

Division 4.

Case 139.—Right disc: Lower nasal margin of disc is a little blurred. Stippling and pigment disturbances of retina are present opposite this area. Vessels appear normal. Left eye: Pronounced physiological cup.

Case 251.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Media normal. Fundi: All details of discs clear. Following the arterioles and venules in their course there are areas in which their outlines become less clearly seen. There is stippling of the retinæ, and small pale cloudy areas in the deeper layers of right and left retinæ towards the periphery, of recent origin.

Case 555.—Some thickening of perivascular lymph sheaths of retinal vessels (right and left). Slight blurring of disc margin but no papillary œdema.

Case 560.—Left disc pale. Venules and perivascular lymph sheaths "plus." Right disc margin blurred; vessels over-full; perivascular lymph sheaths "plus."

Case 596.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Perivascular lymph spaces and venules "plus." Details of margins of right and left discs are not distinct. A clearing-up neuritis.

Case 598.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Discs right and left are hyperæmic; venules dilated and perivascular lymph sheaths near the disc are a little more pronounced than normal. No papillary œdema.

Case 690.—An unusual condition. Increased fulness and width of perivascular lymph sheaths of vessels.

Case 990.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. There is an increase in the perivascular lymph sheaths in right and left fundi. Nothing otherwise abnormal to be seen.

Case 1056.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Commencing stippling of right and left fundi peripherally. Veins a little "plus" and tortuous. No papillary œdema. No blurring of disc margin.

Division 5.

Case 384.—Right vision $\frac{6}{18}$. Left vision $\frac{6}{18}$. Hypermetropic astigmatism. With glasses: right vision $\frac{6}{6}$; left vision $\frac{6}{6}$. At extreme peripheries are to be seen post-retinitic changes in the deep retinal layers. Otherwise nothing abnormal detected.

Case 607.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Venules of right and left fundi are over-distended.

Case 652.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Stippling of left fundus near the papilla. Venules and lymph channels are "plus" in area immediately around the left papilla. Right eye: Nothing abnormal detected.

Case 840.—Blurring of details of elements of discs right and left. Left: stippling and thinning of choroido-retinal coat at temporal margins of disc. Venules and retina otherwise normal.

Case 1061.—Media, clear. Fundi: Some halo about the retinal area around the discs, vessels otherwise normal (? luetic halo). Fovea normal; fields normal.

Case 1274.—Distinct evidence of retinitis. Details of papillæ margins a little obscured.

Division 6.

Case 520.—Venules "plus" in left fundus.

Case 619.—White area in left disc extending half way around the marginal zone. Some fulness of perivascular lymph sheaths. Nothing else abnormal in fundi. Nineteen weeks later no change in fundi.

Case 792.—Vitreous, normal right and left. Both discs are a little hyperæmic, and their margins a little blurred with pigment disturbance in the deep layers of the retina. Venules: right normal in size, left "plus." No exudate; no papillary œdema; no stippling.

Case 829.—Fundi are both dark. No stippling. Right: areas of increased lymph sheaths, but venules normal. Left: the same, but sclerosis of retinal arterioles; the condition is not however symmetrical in extent.

Case 969.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Musculature and media nothing abnormal detected. Right and left foveæ, papillæ and fundi, central and peripheral are normal. The superior retinal arterioles are tortuous and there is over-fulness of lymph sheaths round these vessels.

Case 1020.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Media normal. Fields and colour perception are normal. Fundi: discs normal but probably pale in contrast to the man's colour scheme represented in the eyes by deep pigmentation of retina and choroid. There is abnormal size and distribution of perivascular lymph sheaths and there are a few stipplings and rounded cloudy areas in the deeper layers of the retina, especially in the right eye, about four disc diameters in the first fork of the superior (retinal) temporal arterioles.

Case 1032.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. There is some indistinctness about the nasal edge of left disc and the surrounding choroid. Venules are "plus"; otherwise nothing abnormal detected.

Case 1059.—(A reinfection case, having had syphilis three years previously.) Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Both papillæ are abnormal, especially the right, which shows traces of old papillitis not of recent origin. The vessels of the retina have an abnormal increase of perivascular lymph sheaths which is recent. Scattered over right and left fundi are tiny masses of pigment in the deeper layers of the retina and in places a few dots of fatty degeneration.

Case 1064.—Retinal venules are hyperæmic. Blurring of edges of discs.

Case 1084.—Details of both discs quite clear, but there is a halo in the retina around the discs, especially the right, probably indicative of old-standing changes in the lymph paths. A few of the arterioles also have this halo-like area, especially in the periphery. Some pigment cell degeneration in the retina but no marked stippling, choroiditic or post-choroido-retinitic atrophy.

Case 1142.—Chronic choroido-retinitis.

Case 1177.—Right vision $\frac{6}{24}$. Left vision $\frac{6}{6}$. Amblyopia and hypermetropic astigmatism. Musculature normal. Media, normal. Fundi, stippling; no increase of perivascular lymph sheaths; venules, "plus."

Case 1186.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Both retinæ hyperæmic and venules "plus." Outlines of disc margins blurred, but details clear at depths of optic cups right and left. No peripheral retinitis.

Case 1281.—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Media clear. There is some blurring of details at depth of right papilla; venules are "plus," but there is no exudate nor increase in perivascular lymph sheaths. Details of left papilla are almost undimmed, venules are "plus."

Case 1301—Right vision $\frac{6}{6}$. Left vision $\frac{6}{6}$. Media clear. Papillæ details clear. Finest twigs of arterioles show increased perivascular sheaths. Stippling of retina, especially in periphery right and left.

Case 1303.—Right vision $\frac{6}{12}$. Left vision $\frac{6}{6}$. Media, clear. Outlines of papillæ are seen distinctly except in right eye, where there is some exudate along the larger arterioles in the neighbourhood of papilla; venules not enlarged. Left fundus: one small patch of exudate above.

Case 1322.—Media, clear. Papillæ, normal. Stippling of retinæ. In both fundi there is marked increase in perivascular lymph sheaths.

(5) *A Comparison between the Findings from the Examination of the Cerebrospinal Fluid, Nervous System, Ears and Eyes.*

For convenience in comparison we append below a table of all abnormal cases in which at least one of the special examinations has been carried out. A list of all the cases described in detail is given on p. 300.

TABLE SHOWING THE VARIOUS FINDINGS IN EVERY CASE WITH AN ABNORMAL CEREBRO-SPINAL FLUID IN WHICH THE NERVOUS SYSTEM, EARS OR EYES WERE EXAMINED.

Case No.	Time between infection and examination	Cells per cubic millimetre in C.S.F.	Wassermann in C.S.F.	Symptoms of C.N.S. disease	Clinical signs of C.N.S. disease	Ears	Eyes
Division 1.							
845	9 weeks	16	0	0	0	0	—
1241	8 ,,	10	0	0	0	0	—
Division 2.							
513	14 weeks	84	2000	—	—	—	P
519	9 ,,	11	0	0	0	—	—
828	17 ,,	12	0	0	0	0	—
897	6 ,,	10	0	0	0	0	—
936	3 ,,	10	0	0	0	P	—
947	8 ,,	14	?	0	0	P	—
986	9 ,,	186	0000	0	0	0	P
1006	20 ,,	10	0	0	0	0	—
1009	4 ,,	36	0	0	0	0	—
1090	20 ,,	14	0	0	0	0	—
1135	20 ,,	476	0000	0	0	0	P
1163	22 ,,	10	0	0	0	0	—
Division 3.							
593	11 weeks	742	4300	0	0	—	P
645	3 ,,	10	0	0	0	P	P
673	8 ,,	82	3000	—	—	—	0
734	9 ,,	57	0	0	0	—	0
765	10 ,,	24	0	0	0	P	—
770	10 ,,	14	0	0	0	0	—
821	4 ,,	10	0	0	0	P	—
831	11 ,,	10	0	0	0	0	—
856	4 ,,	50	0	0	0	0	P
874	3 ,,	28	0	0	0	0	—
1055	6 ,,	15	0	0	0	0	0
1119	9 ,,	22	0	—	—	—	P
1196	6 ,,	28	0	0	0	0	—
1214	8 ,,	10	0	0	0	0	—
1230	8 ,,	16	0	P	0	P	—
1258	5 ,,	36	4---	0	0	0	P
Division 4.							
189	18 weeks	254	4---	P	0	0	P
251	22 ,,	362	4400	0	0	P	P
548	22 ,,	1000	4400	P	0	—	0
555	25 ,,	894	4000	P	0	—	P
560	16 ,,	834	4100	0	P	P	P
596	14 ,,	10	0	—	—	P	P
598	15 ,,	26	0	0	0	—	P
690	19 ,,	28	0	0	0	P	P
732	16 ,,	48	0	0	0	—	—
744	16 ,,	50	4---	0	0	P	—
780	18 ,,	14	0	0	0	0	—
783	18 ,,	23	0000	0	0	0	—
842	13 ,,	11	0	0	0	0	—
847	14 ,,	12	0	0	0	0	—
870	14 ,,	22	0	0	0	0	—
958	20 ,,	78	0	P	0	0	0
985	14 ,,	22	0000	0	0	—	—
990	22 ,,	80	0000	0	0	0	P
1017	21 ,,	44	0000	0	0	P	0
1021	20 ,,	12	0	0	0	0	—
1056	22 ,,	56	4400	P	P	P	P
1082	22 ,,	10	0	0	0	0	—
1098	22 ,,	140	1000	P	0	0	—
1331	?	34	0	0	0	0	0

TABLE SHOWING THE VARIOUS FINDINGS—*continued.*

Division 5.

Case No.	Time between infection and examination	Cells per cubic millimetre in C.S.F.	Wassermann in C.S.F.	Symptoms of C.N.S. disease	Clinical signs of C.N.S. disease	Ears	Eyes
88	11 years	0	4---	—	—	P	—
384	9 ,,	144	4400	P	P	0	P
607	4½ ,,	38	0	P	0	—	P
652	30 weeks	72	4400	0	0	—	P
726	Denied	66	4400	0	0	—	—
781	7 months	34	0	0	0	0	—
840	25 years	42	0	P	P	0	P
878	15 months	20	0	0	0	P	—
930	18 ,,	22	0	0	0	0	—
956	22 years	10	0	P	0	0	—
1061	10 ,,	32	0	0	0	0	P
1071	7 months	164	0000	—	P	P	—
1274	15 years	200	4400	0	0	P	P

Division 6.

Case No.	Time between infection and examination	Cells per cubic millimetre in C.S.F.	Wassermann in C.S.F.	Symptoms of C.N.S. disease	Clinical signs of C.N.S. disease	Ears	Eyes
125	2½ years	1500	4---	P	0	—	—
276	7 ,,	74	44--	0	0	0	—
279	11 months	26	1000	0	0	0	—
520	2 years	107	4440	0	0	P	P
619	9 months	156	4400	0	0	P	P
620	5 years	128	4400	0	0	—	—
627	4 ,,	14	4---	P	0	P	0
664	21 months	11	0	0	0	0	—
695	14 years	48	?	0	0	—	—
705	9 months	38	4---	0	0	—	—
713	17 ,,	28	Tube broken	0	0	—	—
753	14 ,,	184	0000	P	0	—	—
767	5 years	132	0	P	0	P	—
792	2½ ,,	380	4440	0	0	?	P
825	15 ,,	26	0	P	P	—	—
829	4 ,,	98	4400	0	0	P	P
907	2¾ ,,	50	0	0	0	0	—
957	18 months	10	0	0	0	0	—
969	?	38	4---	0	0	0	P
1020	14 months	50	4000	0	0	0	P
1031	10 ,,	52	4100	0	0	P	0
1032	16 ,,	22	0	0	0	0	P
1059	?	42	4400	0	0	0	P
1064	Denied	16	0	P	—	0	P
1084	3 years	170	4400	P	0	P	P
1130	?	18	4---	0	0	0	—
1137	11 years	92	4400	—	P	—	—
1141	14 ,,	104	4440	P	0	P	0
1142	10 ,,	100	4400	P	P	—	P
1164	7 months	1100	4410	0	0	P	0
1177	2 years	182	4400	0	0	0	P
1186	9 ,,	226	4444	—	—	—	P
1188	8 months	208	4000	0	0	P	0
1281	?	121	4---	0	0	0	P
1301	2 years	20	0	0	0	P	P
1303	17 months	184	4400	0	0	P	P
1322	15 ,,	114	4400	0	0	P	P

TABLE SHOWING THE FINDINGS IN ALL "DOUBTFUL" CASES WHICH WERE EXAMINED.

Division 1.

Case No.	Time between infection and examination	Cells per cubic millimetre in C.S.F.	Wassermann in C.S.F.	Symptoms of C.N.S. disease	Clinical signs of C.N.S. disease	Ears	Eyes
1195	6 weeks	8	0	0	0	0	—
1219	8 ,,	8	0	0	0	P	—
			Division 2.				
838	11 weeks	8	0	0	0	—	—
935	8 ,,	9	0	0	0	0	—
1013	9 ,,	6	0	0	0	P	—
1014	16 ,,	5	0	0	0	0	—
1157	10 ,,	8	0	0	0	0	—
1261	6 ,,	8	0	0	0	P	—
			Division 3.				
771	11 weeks	8	0	P	0	0	—
827	11 ,,	6	0	0	0	0	—
1043	11 ,,	6	0	0	0	0	—
1238	9 ,,	8	0	0	0	P	—
			Division 4.				
835	24 weeks	8	0	0	0	0	—
590	22 ,,	8	0	0	0	—	—
701	17 ,,	8	0	0	0	—	—
934	14 ,,	6	0	0	0	0	—
1007	14 ,,	6	0	0	0	0	—
			Division 5.				
985	10 months	6	0	0	0	0	—
1297	4½ years	8	0	0	0	P	—
			Division 6.				
860	6 years	9	0	0	0	0	—
1175	7 months	8	0	0	0	0	—
1290	21 ,,	8	0	0	0	P	—

V.—THE EFFECT OF TREATMENT UPON THE ABNORMALITIES IN THE CENTRAL NERVOUS SYSTEM.

In fifty-four of the cases which exhibited "doubtful" or "certain" abnormalities in the central nervous system we have been able to repeat the examination of the cerebrospinal fluid on at least one occasion and sometimes more often. All had, of course, received treatment, but it must not necessarily be assumed that any change in the cerebrospinal fluid which is detected is consequent upon such treatment. The abnormalities in the nervous system are so much more common in the early stages of syphilis than are definite nervous manifestations in the later stages, that it must be admitted that many of the original abnormalities clear up more or less spontaneously.

TABLE SHOWING THE DETAILS OF CASES IN WHICH THE CEREBROSPINAL FLUID WAS RE-EXAMINED AFTER TREATMENT.

Case No	BEFORE TREATMENT		Treatment between examinations	Interval (weeks) between first treatment and re-examination	AFTER TREATMENT		Division of disease
	Number of cells	Wassermann reaction			Number of cells	Wassermann reaction	
1219	8	0	3·6 grm. "914"	6	0	0	Division 1
1220	7	0	2·55 ,, ,,	7	4	0	
897	10	0	2·55 ,, ,,	6	0	0	Division 2
1006	10	0	2·05 ,, ,,	12	0	0	
1090	14	0	2·7 ,, ,,	12	24	0000	
1168	10	0	2·7 ,, ,,	5	2	0	
593	740	4300	4·5 ,, ,,	5	20	0	Division 3
			0·75 ,, ,,	9	2	0	
771	8	0	2·7 ,, ,,	7	4	0	
821	10	0	2·55 ,, ,,	14	4	0	
881	10	0	2·7 ,, ,,	8	28	0	
			2·7 ,, ,,	13	12	0	
856	50	0	2·55 ,, ,,	8	5	0	
1043	6	0	2 7 ,, ,,	8	20	0	
1119	22	0	2·7 ,, ,,	6	6	0	
1258	36	4---	2·7 ,, ,,	10	0	0	
189	254	4---	2·7 ,, ,,	8	14	3000	Division 4
355	8	0	2·7 ,, ,,	8	6	0	
548	1000	4400	4·5 ,, ,,	10	42	0	
			2·7 ,, ,,	28	3	0	
555	894	4000	4·5 ,, ,,	6	16	0	
560	884	4100	3·45 ,, ,,	5	70	0	
782	48	0	2·7 ,, ,,	8	2	0	
783	28	0	2·7 ,, ,,	7	2	0	
1007	6	0	2·7 ,, ,,	8	6	0	
				22	2	0	
1021	12	0	2·7 ,, ,,	9	2	0	
				17	9	0000	
1056	290	4---	2·95 ,, Galyl	4	56	4400	
			4·8 ,, "914"	10	22	4100	
384	144	4400	3·6 ,, ,,	5	68	4400	Division 5
			1·05 ,, ,,	14	20	-0--	
				21	28	4000	
607	38	0	2·7 ,, ,,	6	10	0	
			0·75 ,, ,,	24	10	0000	
726	66	4400	2·7 ,, ,,	7	10	4400	
				12	10	4400	
840	42	0	2·55 ,, ,,	10	8	0	
				24	6	0000	
876	5	0	2·4 ,, ,,	6	0	0	
927	16	0	4·45 ,, ,,	5	8	0	
956	10	0	2·55 ,, ,,	9	12	0	
1274	200	4400	2·7 ,, ,,	6	28	0	
1278	8	0	2·55 ,, ,,	13	8	0	
125	1500	4---	1·8 and				
			0·65 ,, "606"	56	18	----	Division 6
			3·45 ,, "914"	96	10	4300	
276	74	4400	2·7 ,, ,,	13	15	4000	
520	107	4440	4·5 ,, ,,	7	16	4---	
				16	10	0	
619	156	4400	3·3 ,, ,,	19	12	4000	
620	128	4400	2·25 ,, ,,	6	14	0	
627	14	4---	2·7 ,, ,,	8	2	0	
				11	2	0	
664	11	0	2·7 ,, ,,	8	2	0	
705	38	4---	2·7 ,, ,,	6	28	0	
				25	252	4400	

TABLE SHOWING THE DETAILS OF CASES—*continued.*

Case No.	Before Treatment		Treatment between examinations	Interval (weeks) between first treatment and re-examination	After Treatment		Division of disease
	Number of cells	Wassermann reaction			Number of cells	Wassermann reaction	
713	28	----		14	6	0	
740	112	4000	3·3 grm. "914"	6	10	3000	
			3·6 ,, ,,	14	10	3000	
753	184	0	1·8 ,, ,,	11	24	0	
767	192	0	3·15 ,, ,,	8	14	4---	
792	380	4440	2·7 ,, ,,	8	70	4400	
957	10	0	2·7 ,, ,,	10	4	0	
969	38	4---	2·7 ,, ,,	11	16	4---	
1084	170	44--	2·7 ,, ,,	8	26	4441	
1137	92	4400	2·4 ,, ,,	7	6	0	
1141	102	4440	2·4 ,, ,,	5	28	4400	
1164	1100	4410	4·65 ,, ,,	7	30	4000	
			? ,, ,,	10	0	3000	
1177	182	4400		7	8	4000	
			2·7 ,, ,,	13	4	0000	

Doubtful Cases.

Eight cases were examined with negative Wassermann reactions in the cerebrospinal fluids, but with cells numbering 5 to 9 per cubic millimetre inclusive. Of these, four became quite normal after treatment; three remained doubtful, while one (No. 1043 in Division 3) actually became definitely abnormal.

Case 1043.—Infected January 28, 1918. Examined April 17, 1918: Acquired phimosis, balanitis, subpreputial sores, marked general adenitis, papular syphilide; *S. pallida* found in penile sores, April 14, 1918; 6 L, W.R. 4/0, N. normal, B. abnormal, O. not examined, S. normal. He had six doses of 0·45 grm. "914" at intervals of three days from April 18 to May 7, 1918. Re-examined on June 13, 1918: 20 L., W.R. 0/0.

It is of interest to note that the disease in the general system was retrogressive as evidenced by the negative reaction in the serum, while the disease in the central nervous system was progressive.

Positive Cases.

Among the positive cases thirteen became quite normal. Most of these were only slightly affected at the beginning, but others had pronounced abnormalities; thus:—

Case 548.—Infected during the first week in September, 1917. Examined January 31, 1918: Scar of indurated sore in coronal sulcus; marked general adenitis; folliculo-papular syphilide; 940 L.+60 P., W.R. 4/4400, N. normal,

B. not examined, O. normal, S. positive (*vide* p. 281). Between January 31 and February 28, 1918, treated with 4·5 grm. "914" in nine doses. Re-examined on March 8: 42 L., W.R. -/0. From March 8 to March 18, 1918, given 2·7 grm. "914" in three doses. Re-examined June 4: 3L, W.R. 0/0. On January 31, 1918, *S. pallida* was found in the cerebrospinal fluid (*vide* p. 281).

Case 1177.—Infected in May, 1916. Had a sore on penis, treated with mercury injections for three months. Examined on May 7, 1918: Scars on glans, 182L., W.R. 4/4400; N. normal, S. normal, O. abnormal (*vide* p. 288) S. none. From May 8 to May 23 treated with 2·7 grm. "914" in six doses. Re-examined June 27, 1918: 8 L., W.R. -/4000. Again re-examined on August 8, 1918: 4 L., W.R. 4/0000.

The majority of the positive cases showed after treatment a marked diminution of the abnormal signs, although during the period of observation they had not become entirely normal.

Case 560.—Infected in October, 1917. Examined March 2, 1918: Scar of sore at frænum: general adenitis; mucous patches on tonsils and in mouth; condylomata ani; 834 L., W.R. 4/4100, N. abnormal (*vide* p. 282), B. abnormal, O. abnormal (*vide* p. 286), S. none. Treated with ten doses of "914," amounting to 4·95 grm., from February 2 to March 22, 1918. On March 9: 70 L., W.R. 0/0.

Case 619.—Infected in May, 1917. Originally had a sore on the penis. No treatment. Examined on February 9, 1918: Gonorrhœa; general adenitis; 156 L., W.R. 4/4400, N. normal, B. abnormal, O. abnormal (*vide* p. 287), S. none. Treated with seven doses of "914," comprising 3·3 grm. from February 9 to March 3, 1918. On June 18: 12 L., W.R. 4/4000. No change in the eye condition.

Case 792.—Infected in September, 1918. Had a sore on the scrotum and a secondary rash. In December and January, 1916-17, he was treated with 1·65 grm. of "914" and twenty-three injections of mercury. The W.R. was positive and became negative in March. In September, 1917, the W.R. relapsed and was also found positive in December, 1917. Examined on March 13, 1918: Scar of sore on scrotum; general adenitis; 380 L., W.R. 4/4440, N. normal, B. ?, O. abnormal (*vide* p. 287), S. none. Treated with 2·7 grm. "914" in six doses between March 13 and March 28. On May 9: 70 L., W.R. -/4400. Again treated with 2·7 grm. "914."

Three of the positive cases showed a definite increase in the abnormal signs after treatment:—

Case 1090.—Infected November, 1917. Said to have had a sore on the glans in January, 1918. Examined April 27, 1918: No scar: slight general adenitis, 14 L., W.R. 4/0, N. normal, B. normal, O. not examined, S. none. Treated with six doses of 0·45 grm. "914" between April 27 and May 13. On July 30, 1918: L. 24, W.R. 4/0.

Case 705.—Infected on April 6, 1917. Examined on April 25, 1917: Sore on glans; *S. pallida* positive. Treated with 1·65 grm. "914" in three doses from April 28 to May 22, 1917. In December, 1917, W.R. found to be positive and also on February 8, 1918. Re-examined on February 14, 1918: Multiple erosions on edge of prepuce and in sulcus; *S. pallida* not found; scars of sores on penis and scrotum; marked general adenitis; 38 L., W.R. 4/4---, N. normal, B. ?, O. not examined, S. none. Treated with 2·7 grm. "914" in six doses between February 14 and March 1, 1918. Re-examined April 3, 1918: 2 L., W.R. -/0. Again re-examined on August 15, 1918: 252 L., W.R. 4/4400. Treated with 2·55 grm. "914" in three doses between August 16 and September 3, 1918. Again re-examined on September 4, 1918: N., B., S., not examined, O. positive.

Case 831.—Infected December 24, 1917. Examined March 13, 1918: Acquired phimosis, subpreputial sores; *S. pallida* not looked for in view of local antiseptic applications; balanitis; general adenitis; papulo-squamous syphilide; gonorrhœa; 10 L., W.R. 4/0, N. normal, B. normal, O. not examined, S. none. Treated with 2·7 grm. "914" in six doses between March 14 and March 29, 1918. Re-examined May 10, 1918: 28 L., W.R. -/0. Treated with 2·7 grm. "914" in four doses between May 11 and May 26. Again re-examined on June 13, 1918: 12 L., W.R. 0/0. On September 13, 1918, he was recalled because the Wassermann reaction in the serum had become positive. On September 18, 1918: 262 L., W.R. 4/4400. No active lesions of syphilis apparent; N. normal, B. slightly diminished on both sides, O. not examined, S. none. He was given further treatment.

With regard to these three cases it will be seen that Case 1090 shows a continuation of the pathological process in the central nervous system in spite of treatment and a continuation of the positive Wassermann reaction in the serum. Case 705 is a case of early primary syphilis somewhat insufficiently treated. He was found later to have a positive Wassermann reaction in the serum and cerebrospinal fluid and numerous erosions on the penis. In view of the negative spirochæte test and the signs generally, it is probable that this indicates a relapse rather than a reinfection. The disease diminished on further treatment, but again relapsed more strongly and the man was then found to have a lesion of the eye. Case 831 illustrates a progression of the pathological process in spite of treatment, which later partially cleared up, and then relapsed to a much more abnormal condition than at the outset.

Negative Cases.

Cases which showed no abnormality at the first examination were not recalled, and therefore we have no evidence that such cases may develop a lesion of the central nervous system after treatment. We have not yet observed an instance of "nerve relapse" in any case in which the central nervous system was originally found to be normal, but such may of course occur later as an accompaniment of a general relapse.

VI.—THE RELATION OF THE WASSERMANN REACTION IN THE SERUM TO THE CONDITION OF THE CENTRAL NERVOUS SYSTEM.

It is of some practical importance to determine whether:—

(1) A negative reaction in the serum excludes a pathological process in the central nervous system.

(2) A persistent positive reaction in the serum indicates a pathological process in the central nervous system.

(1) This question has to be considered from two different points of view, depending upon whether the case is untreated or treated.

Untreated cases of syphilis show in practically every instance a positive reaction in the serum, except when they are observed in the early primary stage. Therefore the only cases which throw light upon the subject under discussion are the Division 1 cases in which the reaction is negative. There are eleven instances of abnormality in Division 1 in our series. In nine the cases were "doubtful," while in two (Cases 845 and 1241) they were abnormal, namely, 10 and 16 cells per cubic millimetre.

It may therefore be held that these two cases of primary syphilis with a negative reaction in the serum have nevertheless a pathological process in the central nervous system.

With regard to *treated* cases, it is and has been well known that certain syphilitic lesions of the central nervous system may develop as a relapse while the serum is still negative. Such lesions are usually affections of the cranial nerves. Anti-syphilitic treatment has undoubtedly a more effective action upon syphilitic disease in the general system, of which the serum reaction is an indicator, than in the cerebrospinal system, and thus spirochætes may be left to relapse in the latter when the former has been sterilized. This is undoubtedly the explanation of the occurrence of relapses in the central nervous system when the serum reaction is negative.

We have observed two instances of this phenomenon in the whole of the present series:—

Case 907.—Infected August 4, 1915. He had gonorrhœa and a sore on the penis. He was treated with one dose of "606" in October, 1915, followed by Hg pills for six months. On March 13, 1918, he was given one dose of galyl. He was examined by us on March 23, and no lesions were present except marked general adenitis. 50 L., W.R. 0/0, N. 0, B. 0, O. not examined, S. none. He was treated with 2·7 grm. "914" from March 24 to April 8. On August 23 the Wassermann reaction in the serum was also negative.

This man, therefore, had a syphilitic meningitis (50 cells) associated with a negative reaction in the serum.

Case 1071.—Infected September 1, 1917. Sore on penis which apparently remained untreated. It healed up and broke down again in January, 1918. *S. pallida* positive. Treated with 0·75 grm. galyl during January and February. Examined by us on April 20, 1918: Facial paralysis and deafness on the left side had developed on April 4; general adenitis; complete facial paralysis and internal ear deafness; 164 L., W.R. 0/0000, N. abnormal (*vide* p. 282), B. diminished on both sides, right > left, O. not examined, S. (*vide* p. 282). Refused more than four doses of "914." On May 5 the W.R. was positive in the serum.

This is a typical instance of a "nerve relapse" associated at first with a negative reaction which later became positive.

Similarly a progressive disease may be present in the central nervous system without producing symptoms, while the serum is found momentarily negative as the result of treatment. Such an example is to be found in Case 1043 described on p. 293. Thus it must be admitted that the fact of a negative reaction in the serum does not exclude a progressive nervous disease, whether the case has been treated or not. Nevertheless it may be said to render the existence of such a nervous disease improbable, but, in view of the importance of early treatment in such cases, it is doubtful whether it is justifiable to withhold the proof of lumbar puncture in any instance. This is of course especially important whenever the central nervous system has been shown already to have been affected previously.

(2) When the Wassermann reaction persists in a positive condition beyond the period when it should normally become negative as the result of treatment, it is to be assumed that a refractory focus of disease exists in the body, and this refractory focus may be in the central nervous system. Our results give some indication that this assumption is often correct. In twenty-six of the cases in which the central nervous system was abnormal and in which repeated examinations of the serum and cerebrospinal fluid were made, no less than fourteen still showed a positive reaction in the serum, when in a "normal" case

it would be expected to be negative. This is a higher proportion than would ordinarily be found in a series of cases with unaffected cerebrospinal systems, and therefore it may be assumed that a positive reaction which persists to an abnormal extent is suggestive of disease in the cerebrospinal system, and is thus an indication for immediate lumbar puncture.

VII.—CONCLUSIONS.

In drawing conclusions from the preceding work, we consider it undesirable to discuss any hypothetical questions relating to the connection between these early conditions and the later manifest signs of syphilis in the central nervous system. We propose to confine ourselves to the facts which we have observed.

We have examined 624 syphilitic men, of whom the great majority were in the early stages of the disease and showed no obvious signs of nervous disease. Of these, 18 per cent. showed undoubted signs of an abnormal condition of the central nervous system (p. 271). Thus we are able to confirm the work of earlier writers, although the number of abnormal cases is considerably less in our series than in those of some others. This is partly due to the fact that cases which we have described as "doubtful" are not included in this estimate. If, as we consider justifiably, these also are looked upon as abnormal, the percentage of affected men reaches 30.

Even this figure is not so high as some found in the literature, and it should be understood at once that statements of the numbers of men affected are totally devoid of significance, unless accompanied by an indication of the *stage of the disease.* We have found that there is a pronounced difference in the incidence of abnormal conditions at different stages of infection (p. 271). There is a progressive increase of these up to the secondary stage (Division 4), but a slight decline in the later manifest secondary and tertiary period (Division 5). In the case of those men, however, in Division 6 who show no symptoms (namely, the so-called latent stage) there is a pronounced increase in the number affected. In our series, of all men who have been affected for more than six months and showed no symptoms, no less than 35 per cent. had abnormal central nervous systems.

If the number of men found to be abnormal by us is less than appears from the work of some other authors, this is adequately explained by the fact that our series includes a large number of men in the first weeks of the disease (p. 272). The onset of the abnormality may be very early. It was even demonstrable in two cases before the Wassermann reaction had become positive in the serum after infection (p. 272). The nature of this abnormality in the central nervous system does not admit of doubt. It is a syphilitic meningitis. We found this opinion not only upon the presence of lymphocytes in the cerebrospinal fluid but also upon the frequent appearance of polymorphonuclear leucocytes in the more pronounced cases. Furthermore, in a large proportion of the abnormal cases, the fluid showed a positive Wassermann reaction (p. 276), while in one case we even found *S. pallida* by the method of dark-ground illumination (p. 280).

There is no evidence that previous arsenical treatment increases either the number or severity of the affected cases (p. 274).

An outstanding feature of these cases is the almost total lack of symptoms. Very few of the men complained of any disability (p. 281) even when the meningitis was acute enough to produce a visible opalescence in the cerebrospinal fluid.

Very few of these men showed any clinical signs of nervous disease detectible by our examination (p. 281). On the other hand a considerable proportion of the abnormal cases had lesions of the eye grounds, which were indeed of slight extent, but which, nevertheless, indicated a commencing retinitis (p. 284).

A certain number of cases showed affections of the internal ears, but there was no evidence that these were commoner than among non-syphilitics or syphilitics with apparently intact nervous systems (p. 283).

Antisyphilitic treatment has a marked ameliorating action upon these conditions. Some are rapidly reduced to normal, while the great majority were shown to be improved within the period of observation (p. 291). On the other hand, a few of the cases showed a remarkable resistance to treatment, indeed the meningitis actually progressed in spite of repeatedly renewed treatment (p. 294).

Although the demonstration of a negative Wassermann reaction in the serum denotes that an affection of the central nervous system is improbable, nevertheless it does not exclude it (p. 296), and in view of the importance of early treatment (and the value of the operation in prognosis) it is probable that lumbar puncture should never be omitted as a routine in the examination of all cases of syphilis.

VIII.—REFERENCES TO CASES QUOTED.

The following cases are described in detail on the pages mentioned :—

Case	88	..	on page	280	Case	907	..	on page	296
,,	251	..	,,	273	,,	936	..	,,	278
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,,	593	..	,,	278	,,	1135	..	,,	280
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,,	673	..	,,	278	,,	1175	..	,,	274
,,	705	..	,,	295	,,	1177	..	,,	294
,,	740	..	,,	278	,,	1195	..	,,	272
,,	771	..	,,	282	,,	1230	..	,,	273
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,,	838	..	,,	273	,,	1297	..	,,	274
,,	845	..	,,	272					

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THE EXISTENCE OF TWO DISTINCT PHYSIOLOGICAL SYSTEMS FOR THE TRANSMISSION OF MOTOR IMPULSES IN PERIPHERAL NERVES.

[A PALEO-KINETIC SYSTEM SUBSERVING THE FUNCTION OF AUTOMATIC AND ASSOCIATED MOVEMENTS; AND A NEO-KINETIC SYSTEM FOR ISOLATED SYNERGIC MOVEMENTS OF CORTICAL ORIGIN.][1]

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CHAPTER I.—INTRODUCTION.

THE problem of motility, notwithstanding the many notable contributions of recent years, is far from a final solution. This is true of reflex action, associated movement, hypertonicity after central lesions, and cerebellar function. It is especially true of all that pertains to the extra-pyramidal motor pathways.

The present study is a preliminary statement of certain views which I hold in regard to these questions.

Some of my conclusions are founded upon clinical evidence only, while others rest upon a firm anatomical basis. Some are merely speculative and of a theoretical nature. Together they constitute a conception of the neural pathways underlying motility which may help to clarify much that is obscure in this important field.

At this time it is not possible for me to pursue my investigations further. My presentation, therefore, is suggestive rather than complete. As the subject, however, is closely related to certain aspects of the neurology of war, this outline of my views is offered in the hope that it may be of service to other students in this field.

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[1] Presented at a meeting of the American Neurological Association, May 9, 1918.

In our conception of motility as it relates to the peripheral nervous system, we think of motor nerves as subserving a single function only, the transmission of motor impulses from the anterior horn-cells of the spinal cord to the skeletal muscles. This is the conception of the final common path as enunciated by Sherrington, in which all varieties of motor impulses converging from the various central pathways are supposed to traverse a single physiological system of peripheral motor neurones.

The present study is a consideration of evidence from various sources which would appear to suggest a modification of this view, to the extent of recognizing two great physiological systems for the transmission of motor impulses in peripheral nerves, corresponding to certain cardinal differences in the nature of movements and their relation to special integrative and correlating centres in the central nervous system.

If motility is resolved into its component parts three fundamental types of movement may be recognized corresponding to important stages in the phyletic development of the nervous system.

There are, for example, reflex movements which are very primitive and correspond to different levels of the segmental nervous system; there are automatic and associated movements which are under the control of certain infra-cortical centres, and there are isolated synergic movements of cortical origin. The isolated synergic movement is the highest type of motility yet produced in the course of evolution of the central nervous system, and finds its most complete expression in man.

The motor impulses of cortical origin pass over a special system of fibres known as the pyramidal tracts, the existence of which has been established by evidence from many sources. One of the most striking proofs of the existence of such a system has been furnished by the developmental method which shows this system to be of comparatively recent origin in the phylogenetic sense, and one of the last of the great neurone systems to receive its myelin sheath.

At birth the pyramidal tracts are still shorn of their myelin sheaths, which develop gradually during the early years of infancy. Van Gehuchten [20] called attention to the interesting fact that even as late as the eighth month of fœtal life the pyramidal tracts in the spinal cord contain no axis cylinders which have not descended below the decussation of the pyramids in the medulla oblongata.

It is therefore evident that other tracts in the spinal cord must also subserve a motor function, as an infant born prematurely at the seventh month displays an extraordinary power and diversity of movement.

There must therefore exist in the cerebrospinal axis other groups of fibres subserving the function of motility, and these are now generally recognized as the extra-pyramidal motor system. In this system the rubro-spinal tract of Monakow is perhaps the best known and most important, arising in the red nucleus and traversing the entire length of the spinal cord. Abundant evidence has also been furnished from other sources—clinical, pathological, experimental and comparative—showing the existence of extrapyramidal motor tracts in the central nervous system; and that these are of especial importance in the lower forms of life where cortical development is rudimentary.

In previous studies I have presented evidence which would appear to indicate that the extrapyramidal motor system is under the control of the corpus striatum and that a loss of the large motor cells of the corpus striatum in man is associated with the clinical phenomena of paralysis agitans. Paralysis agitans, according to this conception, is produced by a break in the strio-spinal system of motility and is the counterpart in the extrapyramidal system of spastic paralysis following lesions of the pyramidal tracts.

In paralysis agitans there is a progressive loss of certain automatic and associated movements, in contrast to spastic paralysis where the isolated synergic movements of cortical origin are abolished. These two systems are therefore anatomically and physiologically distinct, each contributing in its own way to the normal harmony of movement.

The strio-spinal system is the older and more primitive mechanism and the homologue in man of the basal fore-brain bundle and the primary motor fasciculus of the lower forms of life. Automatic and associated movements must therefore be phylogenetically older than the isolated synergic movements of cortical origin, just as reflex movements must have antedated the appearance of automatic and associated movements.

Typical reflex movements are therefore purely spinal and the first to appear in the evolution of animal life. Next in order would come the various automatic and associated movements which would correspond to the development of a primitive correlating mechanism, the corpus striatum. The most recent addition to motility is the isolated synergic movement of cortical origin which permits of the special dissociations underlying the various skeletal acts which have contributed so largely to the progress of man.

Each of these fundamental types of movement is represented and plays an important rôle in motility—all are united and fused by the integrative action of the nervous system. These various mechanisms may, however, be unmasked by the accidents of disease, which allow us to retrace the various evolutionary steps of differentiation and integration of the nervous system.

In the present study evidence is presented which would appear to indicate the existence of a similar physiological division of motor systems in peripheral nerves; one of these is for the transmission of automatic and associated movements, which because of its age and more primitive function may be termed the paleo-kinetic system. The other is for the transmission of isolated synergic movements of cortical origin, and may be distinguished as the neo-kinetic system.

The paleo-kinetic function represents a more primitive and more diffuse form of movement, which is under the control of the corpus striatum and is subserved by a strio-spino-neural system. The neo-kinetic function is a more highly specialized form of movement which is under the control of the cerebral cortex and is subserved by a cortico-spino-neural system.

According to this conception the skeletal musculature which is the peripheral organ of motility is innervated by two distinct systems of fibres which are physiologically and anatomically distinct: A paleo-kinetic system for automatic and associated movements and a neo-kinetic system for the isolated synergic movements of cortical origin.

With this differentiation in the central and peripheral motor system, which represents two distinct phases in the evolutionary development of motility, there is, I believe, a corresponding differentiation of reflex function, so that it will be possible to recognize certain paleo-kinetic and neo-kinetic reflex manifestations referable to one or the other of these two systems.

It is not without interest to recall that Henry Head [6] and his collaborators in their exhaustive study of the afferent nervous system have reached somewhat similar conclusions.

They find evidence in the peripheral nervous system of the existence of two distinct types of sensibility, a protopathic and an epicritic. The protopathic is a lower order of sensation in contrast to the epicritic sensibility which finds its interpretation in the cerebral cortex.

The diffuse primitive character of the protopathic sensibility suggests a certain analogy to the diffuse contractions of automatic and associated movements. On the other hand, the discriminative quality of the epicritic sensibility is in the sensory sphere the counterpart of the isolated synergic movement of cortical origin.

It is also significant that the optic thalamus, which is the great infracortical station for protopathic sensibility, stands at the same physiological level as the corpus striatum which, according to my conception, is the organ for control of automatic and associated movement.

There is accordingly a certain harmony in the conception of the afferent nervous system as enunciated by Henry Head and that of the efferent systems which I shall endeavour to present in the following pages. According to both hypotheses there exists a more primitive system subserving an ancient function which is related to the basal ganglia in addition to a more highly specialized system of cortical origin.

CHAPTER II.—THE RESTORATION OF MOTOR FUNCTION AFTER INJURY TO PERIPHERAL NERVES.

Peripheral nerves in contrast to the central nerve tracts may under suitable conditions regenerate after injury, with more or less complete restoration of function. This regenerative capacity is probably closely related to the existence of a special sheath termed the neurilemma. Generally speaking, sensory function is restored before motor function and if the hypothesis of Henry Head is accepted the primitive or protopathic type of sensibility precedes the return of the more highly specialized epicritic sensation.

There is, however, clinical evidence to show that in the restoration of motor function after injury a certain differentiation of function is also possible and that motility of a more primitive type as represented by automatic and associated movements makes its appearance before the return of the dissociated synergic movements of cortical origin. Thus there is a certain parallelism in the functional restoration of both motor and sensory nerves which appears to be dependent upon the existence of nerve fibres presenting different degrees of vulnerability and specialization.

It has been noted by a number of observers that after injuries of peripheral nerves there is a return of muscle tonus and a diminution of the atrophy long before voluntary movements are in evidence.

Tinel [19] in his treatise on the Injuries of Nerves states that the sequence of events in the regeneration of motor nerves is as follows:

(1) "The first evidence of beginning restoration of function is the return of muscle tone. The muscle develops a firmer consistence and there is a progressive modification of the paralytic posture of the limb. Atrophy also diminishes.

(2) Soon there is a return of muscle sensibility.

(3) The return of voluntary movement is the latest "manifestation of regeneration. It first shows itself by a slight hardening of the muscles which accompanies the efforts of the patient, and particularly the energetic contraction of the antagonistic muscles; no movement, however, is apparent."

"Then there appear faint voluntary movements necessitating considerable effort on the part of the patient and bringing into play the violent synergic contraction of all the muscles of the limb. Finally, the movement becomes definite, but they long remain weak, clumsy and quickly exhausted, evidencing rapid fatigue accompanied by slight trembling."

In this description, which is based on a large experience with gunshot injuries of nerves, it would appear that the earliest evidences of motility in the paralysed distribution are associated with forcible contraction of all the muscles of the limb, viz., associated movements, and that only later do the movements become dissociated and then are feeble and readily exhausted for a considerable period.

Not long since, while serving on a Medical Advisory Board in connection with the draft, there was referred to me the following rather curious case:—

The man was thirty years of age and worked in a tailoring establishment. Fifteen years previously, his right upper arm had been severely gashed in the middle of its posterior aspect by a circular saw in such a manner as to involve the musculospiral nerve. He was taken to a hospital, where the hæmorrhage was checked and the wound sutured. The injury was followed by immediate and complete paralysis in the distribution of the musculospiral nerve. As there was no improvement in the motor function after a year of conservative treatment a secondary operation was performed, and an attempt was made to suture the divided nerve. This operation was performed in Russia and it was impossible to ascertain the condition in which the nerve was found, or the success of the operative procedure.

In the course of time there had taken place a certain improvement in function, but he had never entirely regained the function of the paralysed muscles and the usefulness of the hand was seriously impaired

At the time of my examination, which was fifteen years after the original injury and fourteen years subsequent to the attempted nerve suture, the clinical picture was as follows: There was a ragged scar on the middle of the posterior aspect of the right upper arm which had evidently extended through the muscle mass of the triceps to the bone. In addition, there was the scar of a longitudinal incision on the posterior aspect of the arm in the course of the musculospiral nerve, which coincided with the site of the previous operation.

There was complete paralysis of the supinator longus, and the extensors of the wrist, thumb and fingers for all movements of cortical origin. When the forearm was held out with the hand open, there was the typical position of wrist-drop. Flexion of the elbow-joint produced not the slightest evidence of contraction of the supinator longus. Attempts to extend the thumb, fingers or wrist evoked no corresponding movement of the parts. There was, therefore, a total paralysis for all isolated synergic movements of cortical origin in the distribution of the musculospiral nerve.

When, however, the hand was closed tightly, as in making a fist, all the muscles of the forearm and upper arm were immediately thrown into strong, firm contractions. The supinator longus contracted firmly, and stood out prominently; the extensor muscles on the back of the forearm were felt to contract forcibly and the hand no longer dropped but assumed a position of extension. In this position, palpation showed the long extensors of the thumb and fingers and the extensors of the wrist to be exerting firm contractions.

In other words, muscles which failed to respond to a willed movement of cortical origin were thrown into active contractions when another type of movement was initiated—the grasping movement of the fist belonging to the type of automatic and associated movement in contrast to the isolated extension of the wrist and fingers which is of cortical origin.

On opening the hand, the extensors immediately relaxed, the hand dropped and the attitude and characteristic deformity of musculospiral palsy were again evident.

Sensation, so far as I was able to determine by routine clinical tests, was not affected in the distribution of the radial nerve. The response was prompt to light touch with cotton wool; to pin prick and to heat and cold over the hand and forearm, both anteriorly and posteriorly.

The sense of localization was also present. Pressure over the extensor muscles of the forearm evoked a distinct sensation of pain similar in character and intensity to that of the unaffected side. The sense of posture in the fingers was also intact. If, therefore, a slight disturbance of sensibility was still present it was not detected by usual sensory tests.

On electrical examination, the extensor muscles of the forearm, including the supinator longus, responded promptly to the direct application of both faradic and galvanic currents. There was no sluggishness of the responses and no qualitative changes or reversal of the formula.

I was unable, however, to elicit isolated contractions in the musculospiral distribution with either current on application of the electrode above the site of the injury. Strong currents evoked a general diffuse contraction of all the muscles of the forearm, which was present in the extensors and was especially active in the flexor group.

It was, however, impossible to pick out the musculo-spiral nerve with the electrode and induce the typical extensor movements of the hand and fingers.

How is this curious dissociation of motor activities to be explained? Here was an old injury of the musculospiral nerve in which there was no return of the isolated movements of cortical origin, as, for example, the participation of the supinator longus in flexion of the elbow or the isolated movements of extension of the wrist, thumb and fingers, and yet all of these same muscles would undergo strong contractions on making a fist.

Phylogenetically, the grasping movement of the hand is an older type of movement and is probably closely related to the climbing period of our ancestry. It is, for example, readily demonstrable in the infant before the pyramidal tracts are fully developed. A strong grasping movement of the hands may be observed in infants born prematurely at the seventh month, at which period the axis cylinders of the pyramidal tracts, according to Van Gehuchten, have not yet descended below the decussation and therefore could not be functionating in the spinal cord.

It is therefore reasonable to infer that the various movements of the infant born at the seventh month pass over some motor system other than the pyramidal tract and this mechanism is represented according to my conception by the striospinal system, viz., the corpus striatum and the extra-pyramidal motor tracts. This being an older mechanism for the control of automatic and associated movements, it antedates phylogenetically the corticospinal system: A fact which is corroborated by myelinization studies which show that the ansa system and the antero-lateral ground bundles of the spinal cord receive their myelin sheaths as early as the fifth month of fœtal life.

It would therefore appear that we have in this case of old musculo-spiral palsy a peripheral motor system which is capable of transmitting impulses from the striospinal mechanism, but none for the transmission of the higher and more specialized types of movement of cortical origin.

The injury to the nerve trunk must have been of such a character as to prevent effectually the regeneration of the more highly specialized fibres subserving a cortical function, but owing to the more primitive character of the other motor system regeneration and restoration of function was possible.

Any anatomical differences between these two systems are as yet purely conjectural. The clinical evidence, however, would suggest that the more primitive function is subserved by a lower order of nerve-fibre, possibly non-medullated or very finely medullated nerve-fibres which sprout from the central end of the divided nerve and invade the adjacent parts. Such fibres may bridge a large gap and wander through dense fibrous deposits to reach the distal end of the nerve, and thus subserve the function of primitive motility. Perhaps frequent division of the nerve-fibres may also play a rôle.

Ramon y Cajal [15] and others have shown in studies on the regeneration of nerves that innumerable non-medullated fibres grow out from the central stump and invade the surrounding tissues—many of these fibres undergoing division. It is therefore possible that primitive types of diffuse associated movements are subserved by fibres of this type which are capable of passing through obstructions which would be insuperable to the more highly specialized nerve-fibres subserving a cortical function.

Regeneration studies would appear therefore to favour the idea that the medullary sheath is a factor in the differentiation and specialization of nerve function which permits of the more isolated and dissociated types of cortical movement.

In another patient, a young woman who had suffered a severe compression paralysis of the musculo-spiral nerve, a similar dissociation of motility was observed with return of function. When she first came under my observation, two weeks after the injury, there was complete paralysis of the musculo-spiral nerve, including the supinator longus, the extensors of the wrist, thumb and fingers. The extensor muscles of the forearm were in a state of complete relaxation with typical wrist-drop. There was also anæsthesia to touch, pain and temperature in the radial distribution of the nerve. The muscles showed complete reaction of degeneration. Three months later sensation was practically restored save for paræsthesia and slight impairment of tactile sensibility and localization in the radial distribution. There was, however, no return of power on attempting to carry out isolated movements in the paralysed muscles. Forced flexion of the elbow produced no contraction of the supinator longus, and strong efforts to extend the wrist, thumb, or fingers were of no avail. There was still complete paralysis for isolated synergic movements of cortical origin in the paralysed distribution. When, however, the hand was closed tightly in a grasping movement, as in making a fist, distinct evidence of muscular contractions were palpable both in the supinator longus and the long extensors of the wrist, thumb, and fingers. There was also evident return of muscle tone in the paralysed muscle. The reactions of degeneration were still present.

Here, again, were encountered evidences of a dissociation of motility during the period of restoration of function after injury. There was absolutely no sign of an isolated movement of cortical origin in the paralysed distribution, and yet very evident signs of contraction were produced by a more primitive type of associated movement. This would appear to indicate either a lessened vulnerability of the older system or a more rapid regenerative capacity after injury.

A somewhat similar observation was recorded some years ago by Oppenheim [14]. A 7-year-old girl had a pressure paralysis of the left musculo-spiral nerve of nine weeks' duration. There was wrist-drop and complete paralysis of all the muscles supplied by the musculo-spiral nerve for all forms of voluntary movement. It was noted, however, that when the fingers were tightly closed, as in grasping an object, the hand was thrown into hyperextension. It was also observed that during certain purely automatic acts the hands and fingers were extended. That it was a case of true peripheral palsy was shown by presence of the reactions of degeneration.

Oppenheim attempted an explanation of the phenomena by assuming a loss of cortical memories for movement, a habit paralysis (Gewohnheitslähmung), as this term was used by Huet [7]. He thought that this factor may have been enhanced by the mental condition of the child, who was both imbecile and epileptic. Later, under appropriate electrical treatment, the paralysis cleared up, and cortical movements were completely restored.

The explanation which was offered by Oppenheim, while suggestive, is far from satisfying. It seems hardly possible that cortical memories could have been so completely obliterated after so short a period of time, and why, might one not inquire, is the condition not more frequently encountered after palsies of the peripheral nerves?

It is my belief that the phenomena may be more satisfactorily interpreted by the assumption of the existence of two distinct physiological systems for the transmission of motor impulses in peripheral nerves, and that the paleo-kinetic system is not only less vulnerable, but regenerates more readily after injury. Such a conception would appear to explain quite naturally the early return of automatic and associated movements, which are diffuse and primitive in their nature, while the more highly specialized function of cortical origin would require a longer period for recovery because of the more delicate differentiation of the nerve-fibres subserving the neo-kinetic function.

A somewhat similar manifestation was described by Jacobi as early as 1877, and finds frequent mention in the literature.

Jacobi [12] had suffered in his own person a serious injury of both the musculospiral and median nerves. After recovery he found that it was quite impossible to contract isolated muscle groups, but only all of them together. In other words, diffuse associated contractions were present in the paralysed muscles without, however, the power of isolated movements.

Here, again, in an old lesion of peripheral nerves the strio-spino-neural pathway (paleo-kinetic system) was open, while the cortico-spino-neural pathway (neo-kinetic system) was broken and would not convey impulses from the cerebral cortex to the muscles.

I have also observed a similar dissociation of motility after an old injury of the median nerve, and in a case of old gunshot wound of the external popliteal nerve. In both cases there were distinct evidences of a return of associated movement in the paralysed muscles without the power of isolated movement in the corresponding neural distribution. A return of protopathic sensibility was demonstrable; while the epicritic sensibility was still absent. The paralysed muscles possessed some sense of pain on deep pressure. Electrical examination showed complete reaction of degeneration.

In both cases, exploratory operation revealed serious injury of the nerve trunks, which were embedded in a mass of sclerotic cicatricial tissue.

In these cases, although several years had elapsed since the injury, regeneration of the more highly specialized neo-kinetic system had not taken place and yet paleo-kinetic movements were evidently present, which harmonizes in the sensory sphere with the return of protopathic and the absence of epicritic sensibility. This would therefore appear to confirm my theory that those fibres subserving the more primitive functions of motility traverse with greater ease gaps and sclerotic obstructions than is possible for the more highly differentiated fibres conveying cortical impulses.

The Restoration of Motility after Facial Palsy.

By reason of its rich and comparatively superficial distribution and the character of the muscles innervated, the facial nerve offers peculiar advantages for the study of motor function.

The facial musculature in addition to voluntary movements of cortical origin presents a wide range of movement of another kind, which is concerned with the expression of the emotions. These are essentially of the automatic and associated type.

The centre for the emotional innervation of the face was long held to be localized in the anterior portion of the optic thalamus, following the teachings of Nothnagel and Bechterew.

In previous contributions to the functions of the corpus striatum [8 and 9] I have presented evidence for the assumption that the essential mechanism for the control of the emotional innervation of the face is centred in the corpus striatum and that facial expression is a part of that large system of automatic and associated movements which is under the control of the striatal mechanism.

In paralysis agitans, for example, which is the extreme expression of a loss of the essential motor mechanism of the corpus striatum, a loss of the emotional innervation of the face is one of the most striking symptoms, the Parkinsonian mask.

In such cases, while the face is motionless so far as expression is concerned, there is nevertheless retained the power of cortical innervation (isolated movements) which are simply impaired by the increased tonicity of the musculature. So far, therefore, as facial motility is concerned, there are two great types of movement which may be dissociated by organic disease of the central nervous system: a lesion of the cortico-spinal system (pyramidal tracts), producing spasticity and loss of isolated movement, as in spastic palsies; a lesion of the strio-spinal system (pallidal system) producing rigidity and loss of the automatic and associated movements subserving the function of expression as in paralysis agitans.

The facial muscles have, therefore, a dual function physiologically distinct, and according to my conception this duality of function is subserved peripherally by two distinct systems of nerve-fibres: a strio-neural or paleo-kinetic, and a cortico-neural or neo-kinetic system.

If the restoration of motor function after facial palsy is carefully observed, I think that many facts confirmatory of this point of view will be found.

For example, the first movement to return in cases of facial paralysis is a closure of the eyelid, associated later with a slight contraction of the muscles about the angle of the mouth. This associated movement of the angle of the mouth on closure of the eye becomes more pronounced as improvement continues and antedates often for a considerable time the ability to contract by isolated movement of cortical origin this same region of the face.

Subsequently, as function is restored, other movements of the associated type appear, and more especially in those severe cases in which voluntary activity is long delayed or perhaps permanently affected.

In such cases the affected side becomes contracted by a more or less continual spasm, which, however, is increased and exaggerated by the natural emotional play of the facial muscles in talking, laughing and crying.

Where these conditions obtain it would seem as if the older paleo-kinetic system showed greater powers of regeneration than the more highly specialized neo-kinetic system; hence the diffuse contractural states, hypertonicity and multiplicity of associated movements after old facial palsies.

An interesting case of old facial palsy is cited by Ballance and Stewart [1] in which a contractural condition had developed on the paralysed side, without the power of voluntary movement.

Following an operation for nerve anastomosis, the contractural state disappeared, although the nerve was found completely divided and disconnected, showing that there had been a certain power of innervation, although apparently a complete separation of the divided nerve had taken place.

In the discussion of this case, Ballance says: "This is a condition for which we do not offer any explanation, except possibly in such cases the nerves, though completely disconnected from the brain, do in some way by virtue of the regeneration that has taken place in them exert an influence on the muscles."

According to my conception, such a case would be explained by the passage of a lower order of nerve-fibre of the paleo-kinetic system through the tissues, bridging gaps and overcoming obstructions and eventually reaching the distal portion of the nerve. This regeneration if more primitive would permit a return of muscle tonus and certain automatic and associated movements, the severe nature of the lesion precluding a regeneration of the fibres of the more highly specialized neo-kinetic system. In such an event, the anastomosis operation would naturally have destroyed that small portion of the paleo-kinetic motility which had been restored.

It is perhaps also significant that after cases of nerve anastomosis in which the hypoglossal or spinal accessory nerves have been united with the distal stump of the facial, the emotional expression, as represented by laughter and weeping, remains permanently absent, although a certain power of voluntary control may be attained.

Another interesting group of facts may be elicited by the study of facial paralysis resulting from gradual pressure, as in cases of tumour, either of the temporal bone or of the cerebello-pontine angle.

In such cases of slow compression, it is usually possible to demonstrate some loss of the isolated movements of cortical origin with preservation of automatic and associated movements.

In a patient at present under my observation with a tumour of the petrous portion of the temporal bone a facial palsy has gradually become manifest. Many of the movements of cortical origin, as for example elevation of the brow or isolated contraction of the angle of the mouth, failed to develop on voluntary effort, and yet in the natural emotional play of the facial muscles these same muscle groups were thrown into active contraction. Indeed, it would seem as though there was an emotional hyperactivity in those groups which were paralysed for cortical innervation.

It would almost appear as if the higher cortical mechanism exercised some controlling influence on the lower system, for when this is destroyed the lower system overacts. Perhaps the failure of the cortical impulses to find an outlet on account of the destruction of neo-kinetic fibres lends an added force to the contractions in the paleo-kinetic system. In any event, contractural states and overaction develop with the loss of the cortico-neural system.

I think, therefore, that a careful study of facial palsies will convince most observers that during recovery the automatic and associated movements are the first to appear, and in older cases with imperfect recovery always show a greater restoration of function; furthermore, when the facial nerve is subjected to pressure the neo-kinetic movements are those which are first lost—which would seem to favour the theory of two distinct physiological systems for the transmission of motor impulses, in the cranial as well as the spinal nerves.

CHAPTER III.—ANATOMICAL CONSIDERATIONS.

In the foregoing chapter certain clinical evidence has been presented which would appear to indicate the existence of two distinct physiological systems for the transmission of motor impulses in peripheral nerves. This evidence would also appear to show a cardinal difference in the anatomical structure of these two systems. For the paleo-kinetic system is less vulnerable to the effects of pressure and is the first to show signs of returning function after injury to the nerve. Especial emphasis should also be laid on those remarkable cases in which the paleo-kinetic function is alone demonstrable many years after injury, when there is every reason to assume the existence of a gap or other formidable obstruction to the regenerative activities of the nerve.

All of these facts favour the idea that the paleo-kinetic function is subserved by a lower type of nerve-fibre, as it is well known that a higher differentiation of nerve structure is associated with increased vulnerability to injury and a lessened regenerative capacity.

Are there any facts in the development and finer anatomy of nerve-fibres which might bear correlation with this point of view?

The peripheral nervous system is composed of two types of nerve-fibres—medullated and non-medullated. The non-medullated is similar in structure to the medullated fibre, save for the absence of a myelin sheath. It is composed of an axis cylinder and a neurilemma, and is supposed to subserve the function of the visceral or sympathetic nervous system.

Medullated nerve-fibres vary considerably in size; some are large, others of medium calibre, and many are quite small, not greatly exceeding the diameter of the non-medullated fibre. Those medullated nerve-fibres subserving a motor function terminate in striated musculature by a special nerve-ending or a motor end-plate.

In recent years considerable attention has been given to the presence of another type of end-plate in striated muscles, which is apparently of sympathetic origin, it being the terminal of a non-medullated nerve-fibre (Boeke [2]). It would thus appear that striated muscles are innervated by two distinct systems of fibres, the one medullated and the other of non-medullated type. This theory of a dual innervation of striated muscles as enunciated by Botazzi [3] has received confirmation from other sources, chemical, histological and physiological, and is no doubt destined to play an important rôle in the elucidation of the problem of motility and the various disorders of the neuro-muscular mechanism.

It is held that the anisotropic disk system of muscles which subserves the function of the quick contraction, or the twitch, is supplied by motor cells of the spinal cord, medullated nerve-fibres and motor end-plates. The sarcoplasmic substance, on the other hand, is assumed to be innervated by the sympathetic cells of the cord, the non-medullated nerve-fibres and a special sympathetic motor end-plate.

The sarcoplasm of muscle is that component which is concerned with the slow tonic forms of muscular contractions, and is more plastic in its function.

According to this conception the slow, tonic contraction of muscular tissue is distinct physiologically from that of the quick contraction or the twitch. Langelaan [13] and Sherrington have recently made notable contributions to this subject, and Sherrington [18] has shown the relation existing between the posturing function of muscle tissue and the sarcoplasmic substance in both striated and non-striated muscles.

As the slow plastic component of voluntary muscles is similar to the function of non-striated muscle-fibres of the visceral system which are exclusively innervated by the sympathetic system, it would appear probable that the twitch component is of more recent origin, phylogenetically, and, as it were, has been superadded to the sarcoplasmic substance in the course of evolution in order to allow greater range and variety of movement. If this were true, one might anticipate a greater development of the twitch component as motility became more highly organized and differentiated, and in the isolated movements of cortical origin the sarcostyles or anisotropic disk system would play a greater rôle than in automatic and associated movements.

At the present time opinions are at variance as to the source of this sympathetic innervation of striated muscle-fibres. Certain studies would appear to show that these fibres emerge in the anterior roots, passing in the white ramus to the sympathetic and reaching the muscles by way of the vascular system. In one group of experiments, section of the abdominal sympathetic in cats produced some diminution of muscular tonus. A similar experiment on the dog, however, was not followed by any reduction of tonus; so whatever may be the source of these non-medullated nerve-fibres and sympathetic nerve-endings in striated muscles, the interesting fact is the duality of function which appears now to be well established: the disk system, or sarcostyles, subserving the twitch or quick movement, and the sarcoplasm the more continuous contraction.

It is, I believe, not improbable that certain differences in the functions of the paleo-kinetic and neo-kinetic systems are closely related to this duality of function of the intrinsic muscle mechanism.

The higher differentiation of motility as exemplified in movements of cortical origin being largely dependent upon a greater development and finer dissociation of the anisotropic system, while in many automatic and associated activities, as of the trunk, the postural and tonic element predominates, which is the function of the sarcoplasm.

Non-medullated nerve-fibres in spinal nerves.—Of great interest in relation to the presence of non-medullated nerve-endings in skeletal muscles are the recent researches of Ransom on the histological structure of spinal nerves.

Ransom [16] by the use of a special method of silver impregnation, which he termed the silver-pyradine technique, has been able to demonstrate large numbers of non-medullated nerve-fibres in peripheral nerves. The existence of such fibres in small numbers has long been known, but that they are as numerous as medullated nerve-fibres was not recognized before the publication of Ransom's studies and his special method of staining. In some nerves they may even exceed in number those of the medullated type.

These non-medullated fibres are not gathered on the periphery of the nerve-fibre in small groups as occurs when a twig from the vascular sympathetic joins the nerve-trunk, but are uniformly diffused throughout the whole section of the nerve.

Ransom has also shown that the greater number of these non-medullated fibres pass to the skin and take their origin in the smaller type of ganglion cell in the ganglia of the posterior roots. He states, however, that a few of these fibres also pass to the muscles.

He makes the very interesting suggestion that these fibres, which are evidently of a more primitive type than medullated nerve-fibres, may be concerned with the transmission of protopathic sensibility, which is a more primitive form of sensation and therefore would be related to a more primitive anatomical system in the nerve-trunk. The epicritic sensibility, as it represents a higher and more specialized form of sensory function, would naturally be subserved by a more specialized system of nerve-fibres.

Ransom reached the conclusion that if Head's conception of sensibility is correct, a large proportion of the afferent nerve-fibres of mammals remains in a relatively undifferentiated state and transmit a relatively primitive and diffuse form of sensation, while other afferent fibres have undergone a further differentiation, acquired a myelin sheath and are engaged in the transmission of specialized forms of sensation, including an increased power of localization.

Insulation of the axone by the myelin sheath is probably an important factor in eliminating the element of diffusion so characteristic of primitive sensation.

It would be of great interest to apply Ransom's technique to the question of the paleo-kinetic system in peripheral nerves. Here in the motor sphere is a more primitive and relatively diffused form of motility, which in its regenerative reaction to injury is not unlike that shown by protopathic sensibility. If the existence of a primeval sensory system in peripheral nerves is accepted, on theoretical grounds, there would be good reason for the assumption that a primitive motor system is also present, as both sensory and motor functions must have passed through very similar stages of specialization and phylogeny.

Ventral roots of the spinal cord.—If there exists a neo-kinetic system in peripheral nerves subserving the motor functions of the cortex, such a system would necessarily have a larger representation in the muscles of the upper and lower extremities than in those of the trunk, as the finer cortical movements are chiefly represented in the extremities. This being true one might expect to encounter certain histological differences in the fibres of the anterior roots which would correspond to the relative representation of the paleo-kinetic and neo-kinetic functions.

In the description of the ventral roots of the spinal cord as given in "Quain's Anatomy," the large and medium-sized medullated nerve-fibres are found in greatest number in the cervical and lumbo-sacral roots, the finer fibres constituting only one-sixth of the whole number in these regions.

On the other hand, the finer nerve-fibres preponderate in the thoracic and coccygeal regions in the proportion of three to one. There may, therefore, be some confirmation of my theory in these facts.

For, while the sympathetic fibres of the autonomic system undoubtedly arise at the thoracic and coccygeal levels there are the enormous muscular masses of the trunk to be innervated, and if these are supplied exclusively by medullated nerve-fibres of large or medium calibre, one would hardly expect such a great preponderance of the finer type of nerve-fibre. In this relation it is also of interest to consider the important rôle played by the muscles of the trunk in automatic and associated movements and their comparative unimportance in the finer movements of cortical origin.

Myelinization of the anterior roots.—If my theory is correct that there exists in peripheral nerves a more highly differentiated type of nerve-fibre subserving the neo-kinetic function of the cortex, one might expect such fibres to develop later than those of the paleo-kinetic system and perhaps receive their myelin sheath subsequent to birth as in the case of the pyramidal tracts.

I am aware of no systematic studies covering this field in the peripheral nervous system of man, a method which yielded such fruitful results to Fleichsig in the brain and spinal cord.

There are, however, some interesting investigations in the field of comparative neurology which would appear to show a considerable post-natal myelinization of the anterior roots.

Hatai [5] has made an elaborate study of the number of medullated nerve-fibres in the anterior roots of the white rat at different ages, and has noted a definite increase corresponding to different periods of growth. He reaches the interesting conclusion that "the total number of the medullated fibres in the ventral roots of the spinal nerves increases as the animal becomes older. The rate of increase of these fibres is not the same for the different ages. It is most rapid between the weights of 10·3 and 25·4 grammes (ten to thirty days), after which it becomes slower. The total number at maturity is about 2·7 times that found in the 10·3-gramme rat."

He also states that "at all ages the ventral root near the cord contains more medullated fibres than appear more distally, the second section being taken near the ganglion of the corresponding dorsal root; in other words, the total number of the medullated fibres in the central root decreases from the spinal cord towards the periphery."

It is possible that these findings may bear some correlation with a higher specialization of motor function in peripheral nerves, as the age of myelinization is of paramount importance in indicating the phylogenetic relationship of the various neuronal systems. This is manifested in the corpus striatum, for example, by the early myelinization of the ansa system and in the cerebral cortex by the late development of the pyramidal tracts, and if specialization of function leaves its imprint in this manner on the central nervous system one might anticipate similar results in the peripheral pathways.

Boughton [4] also investigated this question in the oculo-motor nerves of the white rat and has reached somewhat similar conclusions. He states that, "in white rats having body weights of 11 grammes and 414 grammes, there is a nearly regular increase in the number of medullated fibres in the oculo-motor nerves, this increase amounting to 75 per cent."

In this brief sketch of certain histological evidence I would simply indicate in a very general way the possibility of an anatomical basis for these two physiological systems.

It is my belief that they are anatomically distinct, and the proof of my clinical interpretation must await further study along these and other lines, for in the elucidation of finer anatomical problems a knowledge of function must often precede its structural demonstration.

Chapter IV.—The Relation of the Neo-kinetic and Paleo-kinetic Systems to the Classical Types of Central Hypertonus.—Spasticity and Rigidity.

The conception that skeletal muscles are innervated by two physiologically distinct motor systems, subserving paleo-kinetic and neo-kinetic functions of motility, is not without interest in its relation to questions of hypertonus.

Two great types of hypertonicity are recognized; a spastic state which is referable to the cortico-spinal system, and the rigidity of paralysis agitans which I would refer to the strio-spinal pathways [9]. Is there any relationship existing between these central types of muscular rigidity and the paleo-kinetic and neo-kinetic functions of motility?

It is an established fact of physiology that separation of the motor cortex from the lower spinal centres removes a certain restraining influence termed inhibition, which is characterized by a peculiar spasticity of the musculature. If, as I assume, the corpus striatum is the higher regulating and correlating centre of the extrapyramidal motor systems, it should also exercise an inhibitory influence on the lower motor neurones with corresponding evidences of hypertonicity if the restraining influence were removed. Thus both correlating centres, cortical and striatal, would exercise inhibitory control over their respective spinal centres, which, when abolished, would be followed by the phenomena of hypertonus. In the cortico-spinal system this would take the form of spasticity, and in the strio-spinal system that of rigidity—both spasticity and rigidity constituting a hypertonic condition of the musculature, presenting, however, certain distinguishing features.

There are many differences between these two forms of hypertonus which would lend weight to the theory of a paleo-kinetic and neo-kinetic innervation of striated muscles.

These differences in the character of the hypertonicity, I believe, are dependent upon the relative representation of the two intrinsic contractile systems of skeletal muscles—the anisotropic disk system and the sarcoplasm: that of the disk system (sarcostyles) predominating in neo-kinetic motility, and the sarcoplasmic substance playing an important rôle in paleo-kinetic types of motility.

In lower types of movements such as are represented in paleo-kinetic motility there is a large postural or tonic component greater than in isolated synergic movements of cortical origin where a finer individualization of movement is required, with a consequently greater development of the disk function.

In all forms of muscular activity both the tonic and contractile components are represented, but the higher differentiation of motor functions in cortical movement is produced by a predominance of the contractile or quick movement which is dependent upon the sarcostyles of the disk system.

In the higher development of motility, as it is found in the motor cortex, there is a greater utilization of the contractile element as contrasted with the tonic or plastic element in order to attain a more definite and discriminative control of movement.

It would appear as if the higher differentiation of movement is in the direction of dissociation as compared with the more massive associated movements of involuntary character which are largely engaged in walking, running, posture, grasping, &c. In all such motor acts there is an enormous preponderance of massive contractions of a large number of muscles in contrast to the more diversified, disseminated acts of cortical origin; the paleo-kinetic motility is preponderantly sarcoplasmatic, while the neo-kinetic motility has more the contractile character. For example, an extension of the index finger is produced by an isolated contraction of the agonist with simultaneous relaxation of its antagonist. A grasping movement of striatal origin, on the other hand, is characterized by a diffuse massive contraction of practically all the muscles of the hand, forearm and arm, a form of movement which differs fundamentally from a dissociated movement of cortical origin.

Deformity.—One of the most striking results of a lesion of the pyramidal tract is the deformity and attitude of the paralysed members.

In unilateral lesions the attitude corresponds to what has been termed the Wernicke-Mann type of hemiplegia, viz., adduction of the arm, flexion of the elbow, pronation of the hand and flexion of the thumb and fingers. The lower extremity is fixed in extension with overaction of the triceps suræ, producing the pointed toe deformity. The face is paralysed save for those muscles which act symmetrically, as the frontalis and orbicularis palpebrarum, while the emotional innervation is preserved.

A careful study of this form of paralysis with its associated spasticity will, I believe, demonstrate the loss of the neo-kinetic function of motility, the type of the spastic contraction corresponding to the release of the peripheral neo-kinetic systems, or that system of fibres which subserve the function of isolated cortical movements.

A study of a large series of old infantile hemiplegias at the Craig Colony for Epileptics, in which the lesions were essentially cortical, has convinced me of the preservation of the automatic and associated movements on the paralysed side. The paralysis is one purely of neo-kinetic motility. It is for this reason, I believe, that the extremities and chiefly their distal portions, and more especially the hand, show the greatest degree of paralysis and spasticity, as this portion of the musculature presents the highest degree of specialization in the realm of cortical movements. It also explains the relative freedom of the trunk muscles and the root segments of the extremities which are chiefly concerned with the automatic and associated movements of posture, gait, and station.

Contrast, for example, the disturbances of gait, the difficulty in rising from the recumbent or sitting posture, and the paralysis of expression in paralysis agitans and the preponderant involvement of the extremities, and more especially their distal portions in the spastic palsies of cortical origin. While both types of paralysis involve to some extent the whole skeletal musculature, that of the trunk and root segments predominates in the strio-spinal type and the distal portion of the extremities in the cortico-spinal type, due, as I believe, to the character and distribution of the peripheral paleo-kinetic and neo-kinetic systems.

Rigidity.—The rigidity of paralysis agitans differs in its quality and distribution from the spastic state, although presenting the common characteristic of a central hypertonicity.

The increase of tonus is more uniform and of a more waxy character. It is more plastic, and lacks the elastic quality of spasticity—the so-called clasp-knife rigidity—and there are none of the associated symptoms of increased contractility such as clonus and exaggeration of the tendon reflexes.

The deformity and attitude are also quite different: as is evidenced by the slight flexion of the arms and legs, the bent head and body, the mask-like expression of the face, and the interosseal position of the hands. The alteration in attitude and the rigidity of the trunk are very striking features when contrasted with the characteristic deformity of the extremities which characterizes the spastic state.

These peculiarities of the rigidity in paralysis agitans I would refer to the special function of the paleo-kinetic system with its control over automatic and associated movements which are so closely related to gross movements of the extremities and trunk in their relations to posture and progression. Therefore, when the paleo-kinetic system is released from the inhibitory control of the corpus striatum only those peripheral neurones which subserve the paleo-kinetic function of motility are affected with the characteristic distribution of the rigidity and evidence of a relative preponderance of the sarcoplasmic innervation,

The paralysis of this group of cases is one of automatic and associated activities of muscles; and the isolated movements of cortical origin are well preserved, being merely masked by the rigidity of the musculature.

For in this dual innervation of the striated muscles (paleo-kinetic and neo-kinetic) a paralysis of either system, with its resulting hypertonicity, sadly interferes with the functions of the other motor system, which may be quite intact.

In advanced cases in which the hypertonicity has reached a high degree, with fixation of the joints and tendinous structures, the remaining system may be practically useless although retaining its functional power.

In this conception of a dual innervation, I would maintain that each skeletal muscle may be independently contracted through one or the other system, and that the functional loss of either by causing hypertonicity exerts a considerable interference with the function of the remaining system.

Therefore, in the classical types of central hypertonicity, I believe there is strong corroborative evidence of the existence of two separate physiological systems for the transmission of motor impulses in peripheral nerves.

There is a neo-kinetic system for the transmission of isolated movements of cortical origin which has reached its greatest development in the extremities, and especially the human hand. For this reason the spastic contractures are especially pronounced in this portion of the skeletal musculature. The neural control of the anisotropic disk system has reached a higher degree of development in the neo-kinetic system which is evidenced by the clonus, the more elastic quality of the rigidity and the exaggerated tendon reflexes—all indicating a greater development of the contractile element of motility, as contrasted with its more plastic function (sarcoplasmic substance).

The paleo-kinetic system, on the other hand, subserves a lower type of motility which is characterized by automatic and associated movements and is presided over by the corpus striatum. This form of innervation is preponderantly sarcoplasmic which harmonizes with the nature and quality of automatic and associated acts, hence the waxy rigidity of the hypertonus, the characteristic deformity, the absence of clonus and tendon hyperexcitability.

Reflex activities in central types of palsy.—If there exists in the central and peripheral nervous system two great physiological systems of fibres for the transmission of motor impulses of cortical and striatal origin one would expect to encounter certain reflex activities referable to one or the other of these two systems.

The specialization and differentiation of the pathways would be accompanied by a corresponding development and specialization of certain spinal reflex systems—the differentiation of a strio-spinal and cortico-spinal system developing simultaneously with a corresponding spino-neural mechanism. For it would be difficult to conceive of a spinal mechanism which would functionate effectively in the operations of motility without certain of its systems undergoing further development to meet the requirements of the higher central functions. In this way reflex spinal activities would develop which would be related to the striatal and cortical functions and would therefore represent a higher order of spinal mechanism than that of the primitive segmental function of the cord.

What is the state of the reflexes in paralysis agitans and do they shed any light on the existence of a paleo-kinetic system?

First, it is worthy of emphasis that the skin reflexes, e.g., the plantar reflex, the abdominal and cremasteric reflexes, preserve their integrity unchanged until a late period of the disease, when they may suffer some diminution from the purely mechanical conditions of rigid musculature.

This preservation of the skin reflexes I consider of great importance as indicating their probable relationship to the neo-kinetic system. It would appear that the paleo-kinetic system is not concerned with transmission of the impulses underlying the cutaneous reflexes. This form of reflex activity is apparently of a higher type, the afferents being probably related to Head's epicritic system, and the efferents to the central (pyramidal tracts) and peripheral neo-kinetic systems.

The tendon reflexes are also preserved in paralysis agitans and, as a rule, show no special abnormalities. In those types of the disease in which the tremor manifestations predominate, and there is but little rigidity, they are quite frequently exaggerated, but never to the same degree as in purely spastic states.

There are never the associated clonic manifestations, e.g., ankle clonus, patellar and wrist clonus which characterize the spastic type of hypertonicity, and yet the resemblance of the spontaneous rhythmical tremor to the spastic clonus may be mentioned, as I would refer both to the activity of the disk system in contradistinction to the rigidity which is referable to the more plastic and tonic functions of the sarcoplasm.

As rigidity increases with the progress of the disease the tendon reflexes undergo some diminution, due apparently to the increased tonicity of the sarcoplasm. They can, however, usually be elicited, even in a late stage of the affection.

The reflexes in spastic paralysis—After lesions of the cortico-spinal systems (pyramidal tracts) the state of the reflexes is quite different.

The abdominal and cremasteric reflexes are abolished, even when this system is involved in the cerebral cortex itself. This would appear to show that these reflexes have long reflex arcs which pass to the cerebral cortex by way of a cortical afferent system, the efferent impulses then descending the motor tracts to the periphery.

According to my conception these impulses would pass to the cortex by way of Head's epicritic system, and then descend to the pyramidal tracts and the neo-kinetic system in peripheral nerves.

The same is true of the normal plantar reflex. In pyramidal tract disease this is replaced by the Babinski responses which I believe is of striatal or strio-spinal origin. This is an older type of plantar reflex which is present in the newborn, and later is replaced or inhibited by the further development of cortical control.

Other reflexes which are elicitable in pyramidal tract disease, such as the combined flexion of the hip, knee and ankle (Strumpel's tibial phenomenon) I believe are also of striatal origin and reproduce the stepping movement which is one of the automatic associated functions of the corpus striatum—the strio-spinal mechanism.

Very striking is the great exaggeration of the tendon and periosteal reflexes in spastic states and the various clonic manifestations, viz., ankle clonus, patellar and wrist clonus.

These phenomena I would ascribe to the greater activity of the sarcostyles in the isolated movements of cortical origin, so that when released from inhibition there results a greater reflex twitch reaction than when the extrapyramidal system suffers a similar release. It is possible also that the sarcoplasm plays a more subordinate rôle in cortical activities which would allow a greater freedom of the twitch contraction.

It is also not without significance that the exaggeration of reflexes is greatest in the extremities and especially the distal portions, which would correspond to the highest development of cortical movement.

In pyramidal tract disease it is interesting to compare the exquisite exaggeration of the tendon and periosteal reflexes of the hand which represent par excellence the highest cortical function of motility with those elicited from the trunk or cephalic extremities; and also the distribution of the hypertonicity in spastic states to the distribution of the various tendon and periosteal reflexes and their very evident territorial relationship to the muscles subserving the highest functions of cortical motility.

It would therefore appear that in the various reflex activities which accompany palsies of strio-spinal and cortico-spinal origin, there is perhaps also some confirmation of my hypothesis of a paleo-kinetic and neo-kinetic system for the transmission of motor impulses in peripheral nerves.

CHAPTER V.—CONCLUDING REMARKS.

In the evolution of the nervous system progress in the domain of motility is represented by the development of certain fundamental types of movement long recognized by physiologists.

This evolution in the sphere of motion shows very clearly the struggle of the organism to acquire a greater independence of action in relation to the outer world. Simple reflex motility was followed by the more elaborate type of automatic and associated movement and this in turn was succeeded by the highly specialized movements of cortical origin.

Primitive reflex motility is purely spinal and is referable to the various units of the segmental nervous system.

Movements of the isolated synergic type are cortical in origin and are subserved by a cortico-spinal system, the pyramidal tracts.

I have already presented evidence for the assumption that automatic and associated movements are subserved by a strio-spinal system, the controlling mechanism of which is the corpus striatum and the conductive pathways, the extrapyramidal motor tracts.

Thus there are spinal, strio-spinal and cortico-spinal types of motility, all associated and working together harmoniously and representing different stages in the development of the nervous system.

According to the hypothesis formulated in the present study these subdivisions are not confined to the central nervous system but are continued as such in peripheral nerves. The peripheral motor neurones would therefore include two separate and distinct physiological systems related respectively to the strio-spinal and the cortico-spinal systems.

The older of these, the paleo-kinetic system, is engaged in the transmission of the motor impulses of automatic and associated movements, while the more recent neo-kinetic system conveys the motor impulses of cortical origin.

In other words, with the specialization of the central pathways there has been a corresponding specialization and differentiation in peripheral nerves, with the development of separate systems of fibres for striatal and cortical functions. I would therefore recognize a continuous cortico-spino-neural system subserving a neo-kinetic function and a strio-spino-neural system subserving the function of paleo-kinetic motility.

For reasons which have already been expressed I regard it as highly probable that these two systems present anatomical differences which explain their respective reactions to injury—the nerve-fibres of the neo-kinetic system presenting a higher degree of specialization than those of the paleo-kinetic system, thus causing a greater vulnerability and lessened regenerative capacity.

It is, I believe, not without significance that Henry Head has reached somewhat similar conclusions after an exhaustive study of the afferent system, for specialization and differentiation in both the motor and sensory spheres would naturally follow similar pathways in the upward struggle of the organism.

It is my belief that these two motor systems are associated with the development of corresponding reflex activities, so that strio-spinal and cortico-spinal types of reflexes may be recognized. The differentiation of each system is therefore associated with a corresponding differentiation in the spinal mechanism, which may express itself in the form of special forms of spinal reflexes.

It would also appear that certain differences in the two classical types of central hypertonus, spasticity and rigidity—e.g., the distribution and nature of the palsy, the associated deformity, the character of the hypertonicity and the alteration in reflex activity—may be explained on the basis of their relationships to the neo-kinetic and paleo-kinetic systems.

Of special interest is the apparent preponderance of the disk system or sarcostyles in neo-kinetic motility as contrasted with that of the sarcoplasmic function which predominates in paleo-kinetic motility. This suggests that the more highly specialized forms of movement may be associated with an increased functional activity of the disk system.

Much that is now obscure in the occurrence of so-called associated movements following various types of central lesions will, I believe, find their explanation in the conception of a strio-spinal and cortico-spinal system.

Certain associated movements represent simply the return of the original bilaterality of cortical movements which have undergone suppression in the infantile period, while others are frankly related to the striatal mechanism.

The close relationships of the cerebellar function to motility is recognized by all observers. According to the best authorities the cerebellum is an organ which controls muscle tonus and the synergies of movement and is closely related to posture and equilibrium. In recent years, considerable attention has been given to the question of cerebellar localization.

It will, I believe, be fruitful to consider this organ in its possible relation to a paleo-kinetic and neo-kinetic function.

In all forms of motility two components are readily recognized—a kinetic and a static or postural.

The kinetic component is the movement proper, while the static component is that plastic quality of muscle which maintains posture. Posture is present both during rest and during movement, for in every active movement there is a certain stability or postural element which is an essential part of the motion complex.

Here, again, I believe two great functional divisions may be recognized subserving a strio-spinal and a cortico-spinal postural function—the essential organ for the control of which would appear to be the cerebellum.

According to this conception, there is a paleo-cerebellum which controls the postural or static function of automatic and associated movements and a neo-cerebellum for the postural control of isolated synergic movements of cortical origin.

Comparative neurologists have long recognized the existence of a paleo-cerebellum and a neo-cerebellum, the cerebellar hemisphere increasing in size and importance with the development of the cerebral cortex.

The intimate relation of the cerebellar hemispheres of the cerebrum is also shown by the existence of tracts passing between the cerebral cortex and the pontine nuclei which afford direct connexions with the contralateral cerebellar hemisphere.

I would therefore advance the theory that the postural or static component of movement is controlled by the cerebellum and that here also a paleo-kinetic and neo-kinetic function may be recognized, the one standing in relation to the strio-spinal system and the other to the cortico-spinal system.

The paleo-kinetic and the neo-kinetic types of motility would therefore be represented by distinct physiological systems in both the central and peripheral nervous systems, inclusive of the cerebellum.

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OBSERVATIONS ON THE SENSUAL RÔLE OF THE PROPRIOCEPTIVE NERVE-SUPPLY OF THE EXTRINSIC OCULAR MUSCLES.

BY C. S. SHERRINGTON.

THE rich supply of afferent nerve-fibres existent in the muscles which move the eyeball invites inquiry as to what may be the contribution which these nerves make to visual reactions. Their reflex function should, like that of other proprioceptives, relate to the adjustment and maintenance of postures. Ocular posturing, such as the adjustment and maintenance of the gaze, is delicate and almost continual throughout the waking day. And the afferent nerves from muscles, as is known of them from other muscular fields, yield besides their purely reflex taxis of local posture and movement, sensory impressions which are the basis of perceptions of postures and movements, in short subserve muscular sense. Muscular sense being, along with vision, touch, and the "labyrinthine" sense, a chief source whence the mind manufactures sensual "space," the above is tantamount to saying that the proprioceptive nerves of the eyeball muscles may be expected to contribute to space as perceived with the eye. In the literatures of muscular sense and of visual perception one does not however find, at least so far as my acquaintance with those literatures reaches, much advertence to a spatial rôle for the eye-muscle afferents, and if adverted to, such a rôle for them is often not acceded to. For this attitude of negation towards them, two circumstances are probably largely responsible. The spatial endowment of the retinal sense itself is of such pre-eminent and exquisite degree that it has seemed, especially under more recent investigations, a sufficient source in itself for almost every spatial attribute of vision. The likelihood therefore that adjuvant factors of non-retinal origin make any important contribution to visual space has seemed small. Again, the demonstrated existence of a wealth of afferent nerve-fibres in the eye-muscles is a discovery of comparatively recent date. At the time of the studies of visual space-perception by Wells, Panum, Lotze, Aubert, Hering, and others, the possession of muscular sense by the eye muscles, although on general grounds probable, had no proven neurological basis.

Yet, that a sense-datum derived from the external muscles of the eyeball does furnish factors of importance in visual space, even in uniretinal vision, is, I think, demonstrable by extremely simple experiments. Recourse to certain old and often repeated observations seems sufficient to show this, if the findings be reconsidered from the proprioceptive standpoint.

An observation of such a kind is the following: With the head erect let three spots A, B, C, be looked at directly in front, for instance, on a bare wall across the room. Let us suppose one eye only to be used, the other closed. Let the three spots be equally spaced and in one vertical; and suppose the middle one B the point of visual fixation, and at the level of the eye so that the eyeball is in the so-called "primary" position. Suppose A to lie above B and C below B. The spots are perceived as forming one vertical. How is their verticality recognized? An answer often accepted is as follows: The spots stimulate three retinal points α, β, γ, the mental reactions attaching to which give sensual "verticality" as an attribute inherent in the perception of those points taken as a group. Let us test this statement. Without moving the head, let the eye be directed toward three similar spots A′, B′, C′, on some other part of the wall, say higher and to the right, and lying in one vertical, and let the middle one B′ be fixated.

For strict demonstration the plane surface toward which the gaze is directed should, as in the strict demonstration of Listing's law, be perpendicular to the line of vision, which it will not be if the wall is used for the secondary eye-posture as well as for the first. The well-known slight "perspective" deviation then complicates the Listing so-called "false" torsion. A flat screen set perpendicular to the direction of the visual line in the secondary posture chosen for the eye obviates this.

Then these three spots are perceived likewise as situate in one vertical. But Listing's law of the postures of the eyeball tell us, and its demonstration proves to us, that in this second case, although the spot B′ stimulates the same retinal point β as did B, the spot A′ does not stimulate the retinal point α as did A, but a retinal point α' lying to right of α, and the spot C does not stimulate retinal point γ, but a point γ' lying to left of γ.

We have therefore on the above offered explanation a dilemma. The explanation says that an inherent quality attaching to the three retinal points α, β, γ, gives them as a group verticality in visual space. It says the same of three retinal points α', β, γ' which lie in a retinal line inclined to and crossing at β the line formed by α, β, γ.

If it be doubted that the line of retinal points stimulated by spots A′, B′, C′ is really oblique to and crossing the line of retinal points stimulated by A, B, C, we have only to obtain the after-images of A′, B′, C′, and turn the fixation of the eye back to B. The projected after-image of A′ then appears to the left of spot A, and the after-image of C′ to the right of C, and the line connecting the after-images of A′ and C′ runs obliquely through B and appears no longer vertical but at an angle to the vertical. Though seen as vertical when the eyeball was in the secondary position, namely, gazing upwards and to the right, it is now, with the eyeball in the primary position, seen as if it had undergone an anti-clockwise rotation round its middle point. Conversely if the after-images obtained in the primary ocular position from spots A, B, C are projected when spot B′ is fixated, that is with the eyeball in the given secondary position, the line they form no longer appears vertical, but is seen as inclined to and crossing the vertical line formed by A′, B′, C′, as if it had suffered a clockwise rotation round its middle point.

Thus it is clear that verticality is no unalterable inherent sense-datum attaching to the retinal point-group α, β, γ, as such; for, though in one position of the eyeball that group says inalienably "vertical," in another position of the eyeball it says "inclined to the vertical," namely, in this particular case, upwards and to the right. And the like is conversely true of the other line of retinal points α', β, γ'. Indeed, as the ordinary after-image method for demonstration of Listing's law shows, there is no oblique posture of the eyeball in which the after-images belonging to A, B, C, do appear truly vertical. When the gaze is upward to the left the row they form leans up to the left, when the gaze is downward to the right the row leans downward to the right, and when the gaze is down to the left the row leans downward to the left. The inclinations are in fact both in sense and degree those of the well-known findings that demonstrate Listing's law.

The spatial reference, therefore, of a rectilinear row of retinal points, e.g., α, β, γ, says "vertical" *conditionally*. What condition decides it to affirm verticality in some circumstances, non-verticality in others? In other words, on what factors is based its decision discriminating between vertical and non-vertical? For simplicity of statement the discussion may be restricted to the one experiment where, firstly, the vertical row of spots, A, B, C, is fixated, and, secondly, the gaze is then, without other movement than that of the eyeball, turned upward to the right for observing the projected after-images.

The first stage of this experiment may be called obs. 1, the second obs. 2. What is the difference of sensual conditions obtaining between obs. 1 and obs. 2 respectively?

Evidently the only essential difference between them is that the posture of the eyeball has changed. Unless we suppose that the eyeball-muscles, unlike all other striped muscles, are unprovided with muscular sense, the new posture which they have assumed, and given to the eyeball, must be registered and perceptible. And the presumption is high that these muscles are liberally endowed with muscular sense, for they are known to be supplied copiously with proprioceptive nerves—the very system of nerves which in other muscles is the basis of muscular sense. If in an entirely dark room we direct the eyeballs in this direction or in that, we, although without retinal guidance, are still aware of the directions given to the eyeball. And this is so even when the whole front of the eyeball and the inner surface of the lids have been rendered insentient by cocaine. The direction of the eyeball is then judged almost as accurately as with use of the retina in the undarkened room. Such appreciation of the posture of the eyeball must be attributed to muscular sense. The experiment under discussion may therefore be stated thus: In obs. 1 the sensation from the retinal point-group α, β, γ, is accompanied by a muscular-sense impression p from the extrinsic ocular muscles, an impression attributive to their postural state at that time, namely, to the so-called primary position of the eye. In obs. 2 the similarly evoked retinal sensation from the same retinal point group is accompanied by a muscular-sense impression from the eye-muscles which is not p but p^1, attributive to a different postural state obtaining in them, namely, that of the given so-called secondary position assumed by the eyeball in obs. 2. The sensual complex is different, and the resulting perception differs accordingly. Evidently what we might suppose at first acceptance to be a purely visual perception is, in fact, the result of a fusion of retinal sensation with eye-muscle posture-sensation.

Granting this, can there be distinguished in these perceptions, which we may still call visual, the spatial elements which are purely retinal from those to which muscular sense is adjuvant? Let us leave aside, as not germane to the present question, perspective and third-dimensional features, as far as any such attach to these observations, arranged as they are to be as stripped of them as practicable. The spatial features then attaching to the perceptions are briefly as follows: In obs. 1 (1) three spots of a certain size and shape; (2) situate equidistant; (3) on one rectilinear axis; (4) which axis is "vertical," and (5) directly in front of the observer. If any of these features persist from obs. 1, unaltered into obs. 2, we may infer securely that muscular sense of the eyeball muscles is not involved in them. Of the above five features (1) (2) and (3) do continue unaltered in obs. 2; they are, therefore, space-attributes of purely retinal origin. But (4) and (5) have undergone modification in the perception belonging to obs. 2, as compared with that of obs. 1. The row of spots, as seen in obs. 2, is no longer vertical, as it was in obs. 1, but is inclined to the vertical. And in obs. 2 the row is seen not directly in front of the observer, as in obs. 1, but is seen above and to the right. Therefore (4) and (5) are based on fusions of retinal sense with ocular-muscle sense. They may be *called* visual because natively apprehended to be such, but their source is not purely retinal.

A tactual analogue may serve in illustration. Let three points, say the prong-tips of a trifid fork, be set against the skin, e.g., of a finger, by some other person and with one's eyes closed. One can perceive tactually not only that the points are three, and that they lie equidistant in one straight line, but, with approximate correctness, the orientation of that line to the vertical—also, of course, the whereabouts in the field of reach of the hand at which the touch is met. In this case the contribution made to the spatial perception by the muscular sense is well known and no longer debated. The recognition of the whereabouts in the arm's field of reach is based on the muscular sense of the arm, which tells us the arm's posture, e.g., whether outstretched or not, whether outstretched to right or left when the touch is met with, &c. So also with the recognition of the orientation to the vertical of the line of the three touches. Thus, if the line of the prong-tips be set parallel with the finger's long axis, and the finger be vertical when the touches are received, we perceive the line of touches as "vertical." The postural muscle-sense of the arm, including the finger, is here, of course, the factor which gives the touch-row verticality. If the finger be horizontal when the same three skin-points are touched, the touches say "horizontal." Although the touch-loci have not been changed, the concomitant muscle-sense datum—the finger posture—has changed, and the touches now report "horizontally," e.g., of the line of prong-tips, where formerly they reported "vertical."

The analogy between the tactual and the visual results is obvious. Further, the tactual example makes perhaps more evident another sensual factor involved similarly both in its own and in the visual problem, a factor whose consideration was postponed for the time being in the above statement of the two cases. The evaluation of the posture of the arm in relation to surrounding objects is based upon two sets of proprioceptive data. The impulses from the proprioceptive nerves of the limb itself originate the sense-data concerning the posturing of the limb itself, the local posture as it has been termed. But at the same time the proprioceptive nerves of the rest of the body, and especially those of the gravitational labyrinth, are registering the attitude of the whole body and its orientation to the vertical. Perception of the local posture of the limb, e.g., whether its pose is flexion or extension, gives no definite knowledge of its orientation unless there is concurrent perception of the attitude and orientation to the vertical of the rest of the body of which that limb is an appendage. If one leans the trunk to the right, extension of the right arm thrusts the hand toward the ground; if one leans the body to the left, extension of the arm thrusts the hand away from the ground. To orientate, therefore, by a touch with the hand the position in which an object lies, the local muscular sense of the limb is not enough; there must be combined with it the proprioceptive perception of the posture and gravitational orientation of the rest of the body. The previously made statement of the perceiving of the whereabouts of the fork-prongs relatively to oneself has to be amplified as to its factors. In addition to T, the three touches, with purely tactual local signature, and to the sense-datum given by the arms, including the finger's, own posture, there is a third, P, the sense datum of the attitude and gravitational orientation of the rest of the body. So similarly in perceiving whether the row of touches is in a vertical or a horizontal line. The factor p, the local limb posture, says nothing decisive about verticality unless combined with factor P. But in the instance cited above factor P remains unchanged throughout the observation, as also do the three skin-points touched. The only change is p, the posture of the limb, i.e., the finger; the line of the touches is perceived vertical when the finger is vertical, horizontal when the finger is horizontal.

Returning to the visual observations, a like explanation holds. The sense-datum derived from eyeball muscles gives the "local" posture of the eyeball, i.e., the attitude of the eyeball in relation to the head. But in order to evaluate the direction in which the seen object appears the sense-datum p of the "local" posture of the eyeball in the head is not enough. With it must be combined the sense-datum P of the posture and gravitational orientation of the head itself. The sense-datum p is indecisive as to the "direction" from the seeing self of the thing seen, and about the relation of a seen line to verticality, unless it be linked to P. But throughout the instance of visual observation which was chosen for discussion P remained unchanged. The head was fixed and the direction of uniocular gaze was shifted only by shifting in the orbit the local posture of the globus. Also, as said above, the three retinal spots were throughout the observation the same spots. The difference of spatial attributes between the perceptions belonging to the two stages of the observation are therefore due to change in the factor p, i.e., the sense-datum from the ocular muscles, the only factor which does change. The change observed in spatial attribute of the perception was twofold. In the first part of the observation the three spots appear directly in front of oneself, in the second part they appear to the right and above. In the first part they report the line they lie in to be vertical. In the second part they report it inclined, i.e., obliquely upwards to the right. Muscular sense attributive to the extrinsic ocular muscles is therefore a source of certain of the space-attributes of visual perception. It contributes to perceptions of the direction of the regarded object as projected by the observer. It is also one of the factors deciding the apparent verticality of a visual line or of that line's degree of obliquity to the vertical.

Lotze, when introducing the now generally adopted term "local sign," wrote luminously, "by 'local sign' I mean that the position of the stimulated sensual point acts on the mind." The term in its practical application to-day has come, however, to be employed for something more restricted than spatial reference as a pure generality. It is employed for spatial reference to a locus in that combined space-system composed from the so-called spatial senses, e.g., vision, touch, muscular and labyrinthine senses. Indeed, it seems inappropriate to use it for spatial attributes not referred to that common system of so-to-say ordinary workaday sensual space. Henry Watt's theory of a spatial reference constituting the basis of tonal pitch conceives a space reference not orientated in the common workaday space-system of the other senses. In Watt's view tonal pitch is an ordinal arrangement of space-signs giving merely degrees of distance between sensations of auditory modality and so generating impressions of movement but wholly devoid of orientation of position in reference to space perceived by us through other senses. To call the spatial reference in such a case "local sign" seems inadvantageous.

But the space-reference of vision has always orientation in common sensual space. If the position of the stimulated sensual point acts, as Lotze said, on the mind, e.g., in the retina gives visual local sign, it may be expected that where muscular action determines, as with the retina, the position of the stimulated point, a muscular-sense factor will intrude itself into and be, so to say, contained in the "local sign."

Two degrees of visual localization have been long discriminated. The one, localization relative to the fixation point; the other, localization of the fixation point in reference to the observer. The former is often termed "relative" localization, the latter "absolute" localization. The simple observations forming the theme of this paper argue that the principle underlying the difference between these two kinds of local sign in vision is that muscular-sense is a factor in the one and not in the other. That muscular sense is not a factor in relative local sign is tantamount to a recognition that where the space-relation between one sensory point and another cannot be altered by a muscular act, that muscular act contributes nothing sensorial to the perception of the space-relation between the points. The extrinsic muscles of an eyeball move and posture that eyeball's retina as a whole and cannot alter the mutual space-relations between points composing it. In the observations adduced in this paper the sensual line of points retains its intrinsic pattern unmodified by the posturing of the eyeball. The relative local signature of those points is unaltered by altering the muscular sense concomitant in the perceptions. Such local signature may be termed "pure retinal" local signature, because able to be regarded as pertaining to the retina itself purely.

Lotze, it will be remembered, entered upon, indeed raised, the interesting question as to how the retina has come to be endowed with its "relative," or as termed above, "pure" local signature. That question is no part of our problem. But the view he advanced does possibly affect the suitability of the term "pure retinal" signature as equivalent to "relative" local signature. It does so because Lotze attributed the genesis of relative local sign to central sensations of eye-movement and sensations of effort accompanying or forerunning those. He argued that in the retina the distance and direction from the fovea of a peripheral point has become established as a sense-attribute to that point owing to accumulated experience of the degree and kind of central motor discharge necessary for moving the fovea so as to receive the objective image previously received by the peripheral point. He supposed in short that the "relative local sign" attaching to a retinal point has its origin in a long history of experience of muscular sense—not muscular sense as now usually understood with the commotation of peripheral proprioceptive source, but muscular sense in that older and now less prevalent acceptation, implying a "central sensation" gauging the degree and direction of motor discharge. Lotze's view is adverted to here merely to make clear that its appeal to a kind of muscular sense in its attempt to explain the genetic history of "relative" local sign is not touched by the argument we are now following. Hering explicitly denied to muscular sense any share either in relative or in absolute local sign. As regards relative local sign, apart from its possible past genetic history, on which this paper has nothing to submit, the argument of the present paper is in agreement with Hering.

But with regard to absolute local signature the case is different. The principle emerged above that where a muscular act cannot alter the space-relation between two receptive sensory points, its proprioceptive sensation does not affect or enter into the perception of that space-relation. Conversely, where a muscular act does alter the space-relation between two such points its sensation does enter in the perception of that space-relation.

There is a spatial point in the head which Hering termed the "basal point of vision." "The head, or more exactly the place where, with reference to the object viewed, we imagine our head to be, forms the centre of visual perception." The position of this point may be defined as corresponding with the centre of rotation of Hering's cyclopean eye, or briefly paraphrasing, the cyclopean point of outlook. Though a subjective point, it corresponds with a material point; this latter, as Hering showed, lies in the mid-sagittal plane of the head. And in that plane its position is where that plane is intersected by a line joining the centres of rotation of the two eyeballs right and left. The movements and postures executed by the extrinsic ocular muscles affect the spatial relation between their retina and this material point. Therefore, into the visual space-signature which refers to spatial relation between the perceptual object and the point of visual outlook, there will enter the muscular sense attribute of the movements and postures executed by the extrinsic ocular muscles. Hence, in the observations cited above, a change confined to alteration of the posture of the eyeball brings a change in the "absolute" local signature, a change in the spatial reference of the retinal point. The sensation pertaining to the stimulated retinal point is projected to a locus in sensual space surrounding the subjective point of outlook, and that locus shifts with shift in the eyeball's posture. The absolute local signature has therefore in it a muscular sense factor derived from the extrinsic ocular muscles.

There remains obviously a further element in visual space-assignment. Lotze says, were vision our sole sense our visual field would be an expanse without upper or lower or right or left, and for the same reason that no one in thinking of the universe as a whole regards it as being erect or vertical or horizontal, inclined, &c. Yet, in visual perception, among the seemingly elemental features of it are verticality, horizontality, &c. We have to consider on what this orientated nature of visual space is based. In the simple observations, the subject of this paper, when the retina has passed from the primary visual posture to the oblique secondary, and by so doing has arrived at a posture exhibiting the so-called "false-torsion" of the authors, objective verticality is still perceived, although through partly a new line of retinal points. The only other change in the conditions of the observations is the altered muscular state and altered muscular sensation attaching to maintenance of the eyeball in its new posture, namely, the oblique secondary posture.

That is a proprioceptive factor. Now, obvious considerations urge that the main agency by which the environment impresses upon sense its orientation translated by the mind as right-side-upness or deviations therefrom, is gravitation, i.e., the direction of the line of gravity. This seems indeed the only direct environmental influence by which that orientation becomes an immediate sensual datum. It is the only one which in so far as concerns that orientation causes reflexes directly and *per se.* In some fields of musculature gravity undoubtedly affects physically the muscular reactions in considerable measure, although whether reflexes or sensations are thereby excited is as yet insufficiently known. But the muscles of the eyeball constitute a field of proprioception which gravity can hardly affect more than so slightly as to let one suppose its influence there must be of negligible degree. There is, however, an organ, the otic labyrinth, definitely reacting to gravity. We know that organ to be the source of postural reflexes employing the extrinsic eye-muscles. The proprioceptive mechanism of the eyeball muscles is therefore yoked with that of the otic labyrinth in reflex action. Following the principle established in like cases, the inference is that the sense-impressions of the two are likewise associated. This means that the orientation of the retina in space in regard to the line of gravity, that is to say in the mind in regard to verticality, results from co-operation between the special gravity sense-organ and the muscular sense of the orbital muscles. The so-called primary position of the eyeball is therefore a posture in which the extrinsic ocular muscles cooperate with the otic labyrinth, and the perception of that posture is based on sense-data from both those sources. That posture, however, because it is sensed visually, is perceived by the mind not as a state of the eyeball, but as a state of the external world, of the world as seen; it is "projected."

It appears, so far as space orientation is concerned, as a direct perception of the orientation of the external world. In the observation above the verticality of the line seen by the three retinal points in the primary eye-posture has therefore an otic and an ocular muscle factor combined in it. That a change as regards verticality occurs for those same three retinal points when the eye-posture becomes a secondary oblique one, although the head and therefore the labyrinth posture remains unchanged, shows that in the visual perception of verticality the otic labyrinth datum is so bound up with the proprioceptive datum from the eye-muscles that the latter is a condition for the meaning of the former. Visual local signature as regards the spatial orientation connoted by verticality and deviations therefrom is therefore compounded of at least three factors, namely, retinal, proprioceptive from the extensive eye-muscles, and otic labyrinthism. There is almost certainly a further, though probably minor, factor in it, and that a proprioceptive one, with its source in the muscles of the neck. Recent experiments by de Kleijn (*Archives néerlandaises de Physiologie*, tome ii, p. 644, 1918), show that in the rabbit alterations in the posture of the neck, especially of the two uppermost segments, provoke and maintain definite postures of the eyeballs. These he determined after previous extirpation of the otic labyrinths. They resembled those provocable from the labyrinths themselves, and like those latter were, so to say, in the interest of retaining the retina in the position from which the altered posture of the head displaced it. Sensory impressions of neck-posture have therefore probably to be added to the factors implicated in visual perception of orientation as regards verticality and inclinations thereto.

The above simple observations have been confined to uniocular vision in order to render description and consideration of them more easily given and more clearly followed. But the instances cited, though individual cases only, are of widely applicable type. The upshot of similar binocular observations is similar and similarly explicable. In binocular observations the greater intrusion of the third dimension makes the problem more complex but at the same time enhances its interest. All, however, that is attempted here is to call attention to some simple yet cogent evidence of an important contribution made by the proprioceptive nerves of the external ocular muscles even to uniretinal space perception. It is not that such contribution is unexpected. On the contrary it is but what on grounds of analogy with other organs, e.g., the hand, we should expect. In so saying, the word "organ" seems preferable to the word "sense." If we state the analogy as one between visual perceptions and other specially endowed perceptions, such as tactual, we run a risk of possibly obscuring what is one of the objects of this short paper to make clear. For what the paper desires to insist upon is that the spatial perceptions under discussion are not purely visual, but are in fact, though such nomenclature would be cumbrous, visuo-musculo-labyrinthine, and their analogy is with tactuo-musculo-labyrinthine.

Finally, apart from subscribing or not subscribing to the general doctrine of psycho-physical parallelism, there is a conviction which the neurologist studying the taxis of the nervous system cannot but derive. If in the sensation or perception which belongs to a particular act analysis reveals components derived from nerves of two or more sensual species, the taxis, even though subconscious, of the muscles employed in that act, whether movement or posture, will likewise involve the co-operation of those same two or more species of afferent nerve. Hence, in the ocular reactions discussed, impairment of the muscular sense of the eye muscles will impair not only the spatial perceptions, but also the taxis, the spatial reaction, even the subconscious and purely reflex taxis, of the eye-movements and eye-postures normally accompanying the perceptions. The proprioceptive afferents of the eye-muscles at their exit from the central nervous system are, unlike the proprioceptive afferents of most muscles, not separated from the motor fibres. This makes a difficulty for the experimental investigation of the influence which their destruction would have upon the movements and postures of the eye. They cannot for experiment be severed apart from the motor fibres themselves. But in tabes we have a disease which attacks systematically the proprioceptive afferents, and spares at least for long the motor fibres. For the problem touched in the present paper it seems significant that squints, often transient in character, are not infrequently an early feature of tabes.

SOME PRINCIPLES OF NEUROLOGY.[1]

BY HENRY HEAD, M.D., F.R.S.

TO-NIGHT we hold the first meeting of this Section after the close of the greatest war in the history of the world. We have scarcely wakened from the long nightmare in which for more than four years we have lived. As with many of its victims, who came under our care, the horror has been so oppressive that the dream still haunts our first waking hours. We of all members of the community have cause to recognize that this victorious peace has been won by the sacrifice of innumerable young lives; and for us older men the future must be dedicated to making the world a better place for those who are to take up our burden.

It was of these young men I was thinking when I meditated the subject of my Presidential Address. For it is exactly twenty-six years since I had the honour of reading my first paper, on "Pain in Visceral Disease," before the Neurological Society. Looking back over a quarter of a century, it seemed to me that I might employ my discourse most profitably by considering certain principles, which underlie the standpoint of neurology to-day. I am not going to weary you with an historical epitome. In this quarter of a century there have been no startling discoveries; but I believe that to-day the outlook of the younger generation is fundamentally different from that in which we were brought up, and I shall attempt to indicate the direction in which it seems to me the science of neurology is moving.

The cataclysmic events of the last four years have shaken men's belief in the old order, and medicine has not escaped the universal demand for a restatement of current values. The young are looking to us to enunciate the principles on which our teaching is founded. They are not disposed to accept without criticism conventional explanations. They regard with scepticism the statements, current in text-books, that the manifestations of an epileptic convulsion are the expression of "irritation of the cortical centres," or that aphasia is explained by

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[1] Presidential Address delivered before the Section of Neurology of the Royal Society of Medicine on November 14, 1918.

"the destruction of visual, auditory or motor images." Their curiosity is no longer satisfied by attributing the sensory changes, caused by a peripheral nerve injury, to a "paræsthesia" or "hypo-æsthesia." And indeed words of this order are but anodynes to the conscience. They explain nothing; they serve only to put to sleep the salutary feeling, that further investigation is necessary.

Satisfaction with words, that are in many cases nothing more than a restatement of the problem in bastard Latin and Greek, was one of the most remarkable characteristics of the last quarter of a century. No better example could be found than the misuse of the word toxæmia. We are assured that certain diseases are due to this cause; but, although we know much about the pathological changes in the central nervous system, the symptoms they produce, and the course of the disease, the "toxin" is purely hypothetical. Everywhere we find this remarkable tendency to explain the known by the unknown.

Now every student of natural science should retain something of the simplicity of the child. You all remember the story of the Emperor and his new clothes. Two weavers asserted that they had woven a cloth of extreme beauty and fineness; it had, however, one unusual characteristic. It was invisible to those who were stupid, or unworthy of the post they held. Neither the Emperor nor his courtiers could, it is true, see the stuff, but each was anxious not to betray himself, and all were loud in its praises. Royal robes were made of this wonderful material; but, as the procession wound through the streets, a child called out: "Why he has nothing on," and the child's saying passed from mouth to mouth among the crowd. So the young to-day are calling to us to recognize that many of the terms we use so glibly mean nothing. They do not cover the nakedness of our explanations.

This satisfaction with names is apparent in our conceptions of disease. No better example could be found than the customary attitude of clinicians towards syphilis of the central nervous system. Tabes dorsalis and general paralysis of the insane are thus spoken of as "diseases." In reality, there are two factors only in the morbid manifestations produced by syphilis of the nervous system, the character of the infective activity and the site of its anatomical incidence. The *Spirochæta pallida* may attack almost any structure, and the "disease" expresses the functions of the tissues affected. Tabes dorsalis is a convenient term to express the fact, that the principal focus of the destructive process lies in the posterior columns, and "general paralysis" simply indicates that it has fallen mainly on the higher cerebral centres.

Year by year the line, once so firmly drawn between tabes dorsalis and dementia paralytica, grew fainter as the identity of the pathological process underlying the two conditions was recognized. With the invention of the term "tabo-paresis," the boundaries between the two diseases fell. We now know that any nervous function, from the lowest reflexes to the highest intellectual activities, may be disturbed in consequence of syphilitic infection of the nervous system; and it is only by arbitrary selection that these diverse signs and symptoms can be erected into "diseases." Then again "parasyphilis" was supposed to be the manifestation of a process, syphilitic in origin, but not strictly syphilitic in nature. We now believe that "parasyphilis" is nothing more than an anaphylactic reaction in the tissues of the central nervous system, which have been rendered hypersensitive in accordance with their peculiar lymphatic supply. "Parasyphilis" does not differ from "chronic cerebral syphilis" in any essential pathological particular. It is merely a similar reaction in a different tissue. No better example could be given of the law that, in morbid conditions of the nervous system, the form assumed by the disease is determined by the site of the destruction, whilst the course it runs is dependent on the natural history of the pathological process.

During the last few years the attitude of the neurologist has undergone a subtle change, not only to disease, but also to its general effect upon the patient. At one time everyone was seeking for a sign or a syndrome. Happy the man who could discover a new reflex; it did not matter that he left it unexplained, and failed to correlate it with other ancillary functions. It was a "fact," and to it his name was attached.

But the discovery of such a phenomenon is nothing more than the statement of the terms of the problem awaiting solution. To find its explanation we must look to the behaviour of the nervous system as a whole, and observe how its reactions are changed. In many cases, this leads to the consideration of phenomena, which would have been thought frivolous by the older generation. Take, for example, the importance attached to the patient's account of his own sensations, or the diagnostic value now attributed to certain dreams.

In 1901, I described certain mental changes, associated with disease of the heart and lungs, and showed that they formed another aspect of referred visceral pain. This work fell dead; for those who were interested in morbid conditions of the internal organs cared nothing for changes in the mental state of the patient; on the other hand the alienists denied the facts, because of the remoteness of Asylum life from the conditions of Hospital experience. They made no effort to discover, whether such remarkable examples of the dependence of mental states on disturbances of bodily functions were really open to their investigation. To-day, however, this work is falling into its place as a small contribution to the relation of body and mind.

Interest in the functions of the nervous system, as revealed by disease, is steadily growing. The English school has always been essentially physiological, from the days of Marshall Hall onwards; but this growing attention to function is mainly due to the teaching of Hughlings Jackson. He sprang from the physiologists of the middle of the last century, and was deeply imbued with the principle of evolution, not only of structure, but of function. But his contemporaries became increasingly obsessed with the study of anatomical and topographical details. As years passed by, the teaching of the day moved away from Jackson's conception of an evolution of function, still demonstrable in the activities of the nervous system. Finally his doctrines were scarcely comprehensible to those of us who were brought into daily contact with him in the wards, seduced as we were by the glib generalities of more popular teachers. But, when we came to think for ourselves, we found that his conception of a functional hierarchy, in which one form of activity was dominated by another, standing higher in the evolutionary scale, explained much that was otherwise inexplicable in the phenomena of nervous disease. This swing back to the physiological aspect was quickened by the exquisite researches of Sherrington, and the issue of his "Integrative Action of the Nervous System," in 1906, marks an epoch in the progress of neurology.

We then began to appreciate, that the existence of the central nervous system was due to the necessity of producing coherent action in a plurisegmental organism. Out of the primitive materials at her disposal, Nature was forced to develop a mechanism capable of integrating the physiological forces evoked by relations with the external world. As the animal rose in the evolutionary scale, the functions required of the nervous system became more and more complex, and out of the anatomical structures of a lower organism had to be developed a machinery capable of exercising these higher activities.

This was only possible by integrating the crude physiological processes excited by external stimulation, or arising in response to the action of internal forces. A prick excites a universal tendency to withdraw the part exposed to stimulation; it can also evoke a highly organized movement, such as the scratch-reflex, capable of removing the noxious object.

But such reflexes are preordained and admit of no choice. It became necessary, therefore, to develop a controlling mechanism that would permit the animal, either to attack the stimulus locally, or to evade its action by flight. Finally, with the development of the neopallium was given the power of voluntary choice; the reaction was no longer unconscious, but could be preceded by a definite act of discrimination.

This adaptation to a clearly determined end demands a multitude of adjustments, and such integration is the business of the central nervous system. It can only be carried out by utilizing, in a modified form, functions originally developed for a somewhat different purpose. Thus, the withdrawal movement, which forms the basic reaction to pricking the sole of the foot, is controlled by higher centres in the mid-brain and forms the flexor aspect of the stepping reflex. But, when this higher influence is removed, at any rate for a time, as for example by total transection of the spinal cord, the lower reflex becomes manifested in its primitive form as a pure movement of withdrawal, accompanied by evacuation of the bladder and rectum.

Structure is anchored in the past and may express, in its anatomical arrangements, functions which have been transmuted. Many of them have been carried up to centres of more recent origin, whilst others are exercised normally, in a refined form, under the control of those higher in the evolutionary scale. But nothing remains in its old form, unless it is of utility to the organism under some possible conjunction of circumstances.

Thus the afferent impulses from the viscera do not normally enter consciousness. The eupeptic knows nothing of the processes of digestion, beyond a certain gentle sense of well-being. But, under abnormal conditions, afferent impressions from the internal organs are capable of arousing pain and discomfort. When this occurs, one form of this sensory disturbance is referred to the superficial structures of the body and is associated with areas of tenderness, corresponding to a segmental arrangement of the central nervous system. This is not a "hyperæsthesia," but a tendency to react more violently to any stimulus capable of evoking discomfort. The threshold is not materially lowered, but the vehemence of the response is greatly increased.

Now this over-reaction is associated with reflexes, which still reveal their old defensive character. As an answer to visceral pain, the abdominal muscles become tonically contracted, and the leg is drawn up on the affected side. The sufferer is forced to creep into some sheltered place, and to lie curled up, till he either dies or recovers. All capacity to move as a whole is abolished; the reactions of the part affected now dominate the activity of the complete animal. Visceral reflexes, of this order, are antagonistic to general movements, and this is expressed, not only in muscular immobility, but in the mental attitude which accompanies visceral pain.

Pain is the oldest defensive reaction, and potentially painful stimuli are the basis of all primitive reflexes. It is therefore of importance for higher development, that these impulses should be rendered less effective in favour of those impressions which lead to a more general and discriminative response. But, although they are controlled and even abolished, the mechanism underlying the production of pain must remain in full physiological activity, ready to play its part should occasion arise, in the defence of the body against noxious influences.

The functions of the central nervous system are not a palimpsest, where a new text is written over an earlier manuscript, partly erased. The more primitive activities have been profoundly modified by the advent of the new centres, which utilize some of the faculties originally possessed by the older mechanism. In many cases the higher function could not be exercised, without the existence of these lower powers which it dominates and controls.

When, however, the higher mechanism is thrown out of action the functions of the lower centres are free to exhibit their activity unchecked.[1] Jackson insisted that the manifestations, due to a lesion of the central nervous system, must be considered from a negative and a positive aspect. Injury to the pyramidal system was shown, negatively, in the loss of the finer voluntary movements; but the activity of the lower centres, released from control, was evident in the spastic rigidity of the paralysed parts. In the same way, removal of the control, normally exercised by the cortex over the activity of the optic thalamus, leads to the remarkable condition known as thalamic over-reaction; all stimuli capable of evoking discomfort, and in some cases even pleasurable excitation, cause an exaggerated response on the affected half of the body. This is not due to "irritation"; it is due to release. It is the direct consequence of loss of that control normally exercised by the cortex over the activities of the optic thalamus. Once this control is removed by disease, the lower centre reacts unchecked to any stimulus capable of exciting a response.

It has been the custom to call upon a hypothetical "irritation" to explain all such positive manifestations of nervous energy. The violent flexor spasms and the outbursts of excessive sweating, which occur when the spinal cord is injured, have been attributed to this cause. In reality, they are the expression of a diffuse and massive response to stimuli, applied to parts below the level of the lesion; they are manifestations of a primitive mode of reaction, due to removal of control from above. Involuntary movements in hemiplegia, automatic acts following an epileptic attack, the emotional utterances in aphasia, are all examples of the same phenomenon. The condition of sensibility, during the protopathic stage of recovery from a peripheral nerve lesion, is another instance of a primitive mode of reaction, released from control by the tardy restoration of the epicritic mechanism. Normally the massive "all or nothing" response of the more primitive system is held in check by the localized and discriminative sensations, due to excitation of the epicritic end-organs. But, when the one set of afferent impulses is permitted to play on the ultimate sensory centres unchecked by those arising in the higher mechanism, the response becomes massive and diffuse, and extensity becomes of greater import than intensity.

Had we applied Jackson's law, that the functions of the nervous system are integrated on evolutionary principles, neurology would not have made so many excursions into the wilderness. He taught us that a lesion of the cerebral cortex caused disorder of movement, not paralysis of the muscles. This lesson, however, was not applied to the other functions of the cortex. Much time was wasted on topographical localization of various sensory centres, before the nature had been determined of the functions exercised by the cortex. Now we know that, as far as somatic sensibility is concerned, the cortex is responsible for the appreciation of spacial relations, for the power of responding to different intensities of stimulation, and for the capacity to recognize similarity and difference in external objects, brought into contact with the body. The cortical centres are not concerned with the crude appreciation of touch, pain, heat and cold.

Such principles are not only of theoretical importance; they are perpetually thrust before the neurologist in his daily work. Clinical diagnosis is a by-product of scientific investigation. It is impossible to expose every patient to laborious scientific examination, nor would it serve any useful purpose to do so; but the simple tests, employed in the wards, are valueless until they have been calibrated by more elaborate investigations. The man who says he can obtain all the information he wants, in cases of injury to peripheral nerves, by means of a pin and a piece of cotton wool, depends on someone else to teach him the significance of these empirical tests. They have no scientific value, until the data they yield are correlated with results, reached by methods capable of measurement.

The charm of neurology, above all other branches of practical medicine, lies in the way it forces us into daily contact with principles. A knowledge of the structure and functions of the nervous system is necessary to explain the simplest phenomena of disease, and this can be only attained by thinking scientifically.

Let us then consider some of the leading principles, which the candidate of the future must master in the course of his initiation:—

(1) When any level[1] of activity is attacked, the most complex functions, and those which have appeared most recently, are the first to suffer; they are also disturbed to a greater degree, and to a wider extent, than those which are simpler or more inevitable in their expression.

An excellent example of this rule is seen in the effect of a lesion of the sensory cortex on the appreciation of the spacial aspects of an external stimulus. Tests, which demand recognition of movement in three dimensions, are more gravely affected than such a comparatively simple one as localizing the position of the stimulated spot; for, with the precautions we habitually adopt, this is an exploration in one-dimensional space. Division of the spacial aspects of sensation according to one, two and three dimensions is obviously arbitrary; but the tests we apply become more or less complex and difficult according as they demand recognition of relations in space of three, two or one dimensions. Consequently, the records obtained by measuring the extent of passive movement, which can be appreciated in the affected limb, are more gravely and extensively affected than those yielded by our method of testing localization.

(2) The negative manifestations of a lesion appear in terms of the affected level.

Thus, if those parts of the cortex are injured which are associated with speech, the consequences are not an affection of some totally different function, such as visual or auditory images, but are shown in terms of defective speech. The business of a clock is to show the time. Faulty action of the main-spring may cause it to go fast, to go slow, or to stop. But we cannot look at the face of the clock and say "That is strong main-spring time"; all we can say is that the clock is fast. So the negative manifestations of a cortical lesion affecting speech are presented to us in the form of a disturbance of speech, not as a destruction of images.

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[1] The word "level" is employed throughout in a strictly functional sense. Any one anatomical organ of the central nervous system may exercise functions of more than one physiological level. Thus the optic thalamus contains, not only the termination of the fillet, but also the centre for the affective aspects of sensation. For the same reason, a lesion of the post-central cortex not only produces loss of certain sensory faculties, but may also cause hypotonia in the affected parts of the body.

In the same way, morbid conditions at the reflex level appear as changes in the reflexes; a disturbance of cerebellar activities is seen in inco-ordinate movement; whilst an injury to the sensory cortex affects the discriminative aspects of sensation, a purely mental function.

(3) A negative lesion produces positive effects by releasing activities, normally held under control by the functions of the affected level.

I have already given many illustrations of this law. The phenomena of the mass-reflex after injury to the spinal cord and of spastic rigidity in hemiplegia are examples on the motor side; protopathic sensibility and thalamic over-reaction illustrate similar conditions amongst afferent activities.

(4) The functions of the central nervous system have been slowly evolved by a continuous process of development. The methods, by which this gradual progress from lower to higher efficiency has been reached, are still manifest in the phenomena of its normal activity.

Thus the lowest reflexes give the most definite response; there is little or no choice, the answer is inevitable. They are, as Jackson expressed it, "highly organized." But further development of the needs of the animal demanded a reaction that was less fixed; some variation must be permitted. This led finally to the domination of many reflex manifestations by consciousness. What at lower levels appeared a variable response, became, with the progress of evolution, voluntary control.

(5) Integration of function within the nervous system is based on a struggle for expression between many potentially different physiological activities.

Let us consider the following example, drawn from the afferent side. A metal test-tube, containing water at 45° C., normally evokes a sensation of pleasant heat. But it is easy to show that this temperature, suitably applied, stimulates the cold-spots and then appears to be cold.

If, by chance, both the end-organs for the reception of heat and cold are absent, pain is produced. Thus the same temperature is capable of exciting heat, cold and pain; and yet under normal circumstances it evokes a sensation of pleasant warmth. Evidently, the incompatible impulses, due to stimulation of the cold- and pain-spots, are prevented from reaching consciousness.

Such integration occurs at several stages, during the passage of afferent impressions through the central nervous system. Those, alike in their qualitative potentialities, are gathered together, whatever may have been their origin, at the periphery. Thus all impulses capable of arousing pain come together in the spinal cord. In the same way, there are separable paths for those associated with sensations of heat and cold. Finally, on higher physiological levels is fought out the struggle for final dominance, and, of the warring impulses, one group only excites a conscious response.

The processes by which this integration is rendered possible are three in number. First of all those impulses, potentially of a like sensory quality, are gathered together. Secondly, all impulses capable of exciting sensations of a different quality are rejected by the receptors, which guard each functional level. The third method is manifested in the phenomena of adaptation. Water at 30° C. may appear under suitable external conditions to be neither hot nor cold. But, when the hand has been previously soaked in water at 45° C. for some time, a temperature of 30° C. will seem to be cold. Conversely adaptation to water at 15° C. converts 30° C. into a warm stimulus. This power of adaptation to the conditions of the environment, and consequent shifting of the neutral point is one of the most important factors in the mechanics of integration. No stimulus acting over a long period can remain continuously at the same level of efficiency; it leads to a state increasingly favourable to the appearance of the opposite phase of activity. This is the essential condition underlying the tendency to biphasic reaction so characteristic of the central nervous system.

These three processes, acceptance by similarity, rejection by difference, and biphasic dispositions, form the main features of the physiological functions of the central nervous system. They are factors which lead to integration, whether we are dealing with reflexes or with such psychical acts as sensations.

They are interposed between the vital consequences, produced by the impact of a physical stimulus on peripheral end-organs, and the final processes of consciousness. We have no right to speak of psychophysical parallelism; we ought rather to consider on the one hand physiologico-physical responses and on the other psycho-physiological relations.

Thus, between the impact of a physical stimulus on the surface of the body and the movement or sensation it evokes, lie the innumerable reactions of the physiological level. Here the form assumed by the conscious response is prepared to a degree scarcely suspected until recently. Many of the impressions produced by a single external stimulus are incompatible with one another, and some of them must give way in the conflict. But the result of the struggle is not fixed or pre-ordained; it may be reversed by some previous occurrence, which has changed the disposition of the central nervous system. The form assumed by a reflex may be determined by the character of the movement which preceded it, and the phenomena of sensory adaptation depend upon the active influence of the past in present events.

Moreover, the general resistance of the central nervous system to certain impulses varies greatly at different times and in different persons. This is particularly evident in the case of pain. We are all subjected to innumerable painful impressions, which under normal conditions never reach consciousness; but if the resistance, either local or general, is lowered, these impulses may underlie definite sensations of pain.

We neurologists are brought into daily contact with these diverse functional reactions and can watch the processes of evolution, visible in the actual behaviour of the central nervous system. We work in the passage-way between the physical universe and the dwelling-place of the mind. We see the coming and the going, and we alone can record which of the many aspirants has conquered the right to enter or to leave that council chamber of human activities.